MATERNAL AND PERINATAL OUTCOME IN PREGNANCY FOLLOWING RECURRENT PREGNANCY LOSS

Dissertation submitted in partial fulfillment of requirements for

M.D. DEGREE BRANCH II

OBSTETRICS AND GYNAECOLOGY
THANJAVUR MEDICAL COLLEGE
THANJAVUR

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY
CHENNAI

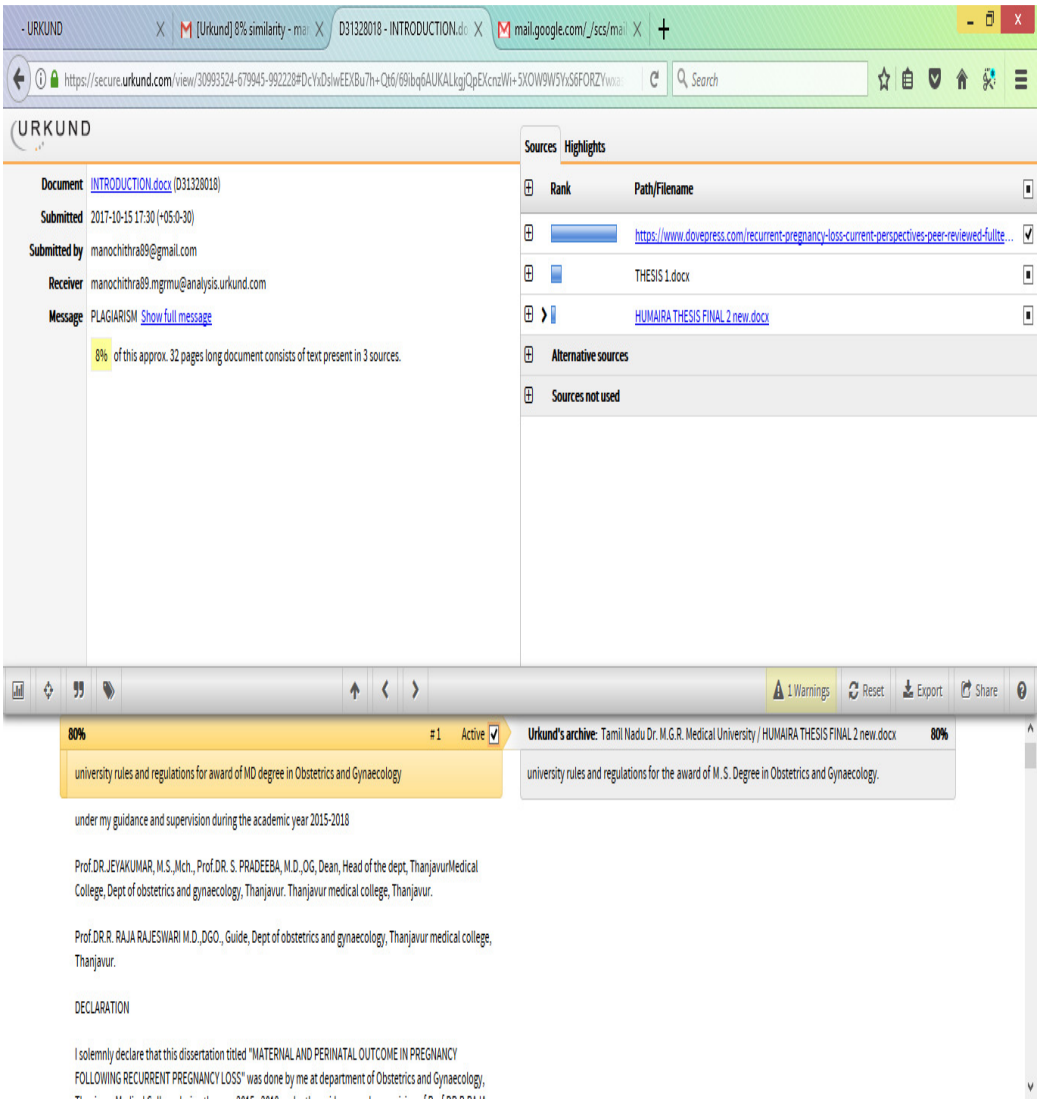
MAY 2018

CERTIFICATE

This is to certify that the dissertation titled " **MATERNAL AND PERINATAL OUTCOME IN PREGNANCY FOLLOWING RECURRENT PREGNANCY LOSS "** is a bonafide work done by **Dr.B.MANOCHITHRA** in the department of Obstetrics and Gynaecology (ThanjavurMedical College) Thanjavur, in partial fulfillment of the university rules and regulations for award of MD degree in Obstetrics and Gynaecology under my guidance and supervision during the academic year 2015-2018

Prof.DR.JEYAKUMAR, M.S.,Mch.,
Dean,
ThanjavurMedical College,
Thanjavur.

Prof.DR. S. PRADEEBA, M.D.,OG,
Guide and Head of the dept,
Dept of obstetrics and gynaecology,
Thanjavur medical college,
Thanjavur.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under my guidance and supervision during the academic year 2015-2018
Prof.DR.JEYAKUMAR, M.S.,Mch., Prof.DR. S. PRADEEBA, M.D.,OG, Dean, Head of the dept, ThanjavurMedical College, Dept of obstetrics and gynaecology, Thanjavur. Thanjavur medical college, Thanjavur.
Prof.DR.R. RAJA RAJESWARI M.D.,DGO., Guide, Dept of obstetrics and gynaecology, Thanjavur medical college, Thanjavur.
DECLARATION
I solemnly declare that this dissertation titled "MATERNAL AND PERINATAL OUTCOME IN PREGNANCY FOLLOWING RECURRENT PREGNANCY LOSS" was done by me at department of Obstetrics and Gynaecology,

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DECLARATION

I solemnly declare that this dissertation titled "MATERNAL AND PERINATAL OUTCOME IN PREGNANCY FOLLOWING RECURRENT PREGNANCY LOSS" was done by me at department of Obstetrics and Gynaecology, Thanjavur Medical College during the year 2015 - 2018 under the guidance and supervision of **Prof.DR.S.PRADEEBA MD OG..,** This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree in Obstetrics and Gynaecology (Branch -II)

Signature of the Candidate

Dr.B.MANOCHITHRA
MD Post Graduate Student
Department of Obstetrics and Gynaecology
Thanjavur Medical College, Thanjavur.

GUIDE: Prof. DR. S. PRADEEBA, M.D.,OG,
HOD, Dept of obstetrics and gynaecology
Thanjavur medical college,
Thanjavur.

Place:

Date:

ACKNOWLEDGEMENT

I gratefully acknowledge and sincerely thank **Prof.Dr.JEYAKUMAR.,M.S.,Mch., The Dean,**Thanjavur Medical College and Hospital, Thanjavur for permitting me to conduct the study and use the facilities of the institution for my study.

I am grateful to the Head of the department, **Prof.Dr.S.PRADEEBA MD.,OG,** department of Obstetrics and Gynaecology, Thanjavur medical college, Thanjavur for being my guide and helping me all through the study.

I sincerely thank **Prof.Dr.R.RAJARAJESWARI MD., DGO.** helping me all through the study. .

I am bound by ties of gratitude to my respected teacher **DR.ANJUPADMASEKAR M.D.,** for her valuable guidance in conducting this study.

I wish to express my sincere thanks to all the Assistant Professors of our department for their support during the study.

I thank the secretary and chairman of Institution Ethical Committee, Thanjavur medical college, Thanjavur.

I also thank **DR. MAHESHWARAN M.D.,** who helped me a lot in doing statistics of my study.

I would be failing in my duty if I don't place my sincere thanks to those patients who were the subjects of my study . Above all I thank God Almighty for His immense blessings.

ABBREVIATIONS

RPL : Recurrent pregnancy loss

hCG : Human chorionic gonadotrophin

PGD : Preimplantation Genetic diagnosis

PGT : Preimplantation Genetic testing

VEGF: Vasculo Endothelial Growth factors

APLA: Antiphospholipid antibody syndrome

LH : Leutinizing Harmone

PCOS: Poly cystic ovarian syndrome

TRH : Thyroxine releasing harmone

PROM: Premature Rupture of membranes

CS : Caeserean Section

IUGR: Intrauterine growth restriction

IUD: Intrauterine fetal death

APH: Antepartum Haemorrhage

CPD: Cephalo Pelvic disproportion

GHT: Gestational Hypertension

CTG: Cardio tocography

AP Eclampsia: Antepartum eclampsia

GDM: Gestational Diabetes Mellitus

NICU: Neonatal intensive Care unit

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INTRODUCTION

Successful reproductive outcome in pregnancy is the goal and motivation of women with a history of recurrent pregnancy loss. Recurrent pregnancy loss (RPL) has a controversial definition.

Recurrent pregnancy loss is a heterogeneous reproductive problem, with multiple aetiologies and contributing factors. As such, evaluating and treating women with this condition is a complex task, and research in the field is no less daunting.

The definition of recurrent pregnancy loss is debated, ranging from two clinical miscarriages, not necessarily consecutive, according to the American Society for Reproductive Medicine (ASRM) (ASRM Practice Committee, 2013) and a joint International Committee for Monitoring Assisted Reproductive Technology and World Health Organization glossary (Zegers-Hochschild et al., 2009), to three consecutive pregnancy losses (not necessarily intrauterine) as defined by both the European Society for Human Reproduction and Embryology (Jauniaux et al., 2006) and the Royal College of Obstetricians and Gynaecologists (RCOG Green Top Guideline, 2011).[1,2,3,4]

Consensus Statement from the ESHRE Special Interest Group, Early Pregnancy (2014) recommend the term recurrent pregnancy loss be used to describe repeated pregnancy demise, and the term recurrent miscarriage be used when all pregnancy losses have been confirmed as intrauterine miscarriages, by ultrasound or histology.[5]

Under normal, non-neoplastic conditions, human chorionic gonadotrophin (hCG) is exclusively produced by the syncytiotrophoblast. It follows logically that a pregnancy loss is the spontaneous demise of a pregnancy, which has been confirmed by at least two positive b-hCGs in the serum or urine (kolte et al, 2014).[6] The general availability of high-resolution transvaginal ultrasound in developed countries, combined with sensitive urine and serum b-hCG measurements, has revolutionized the diagnosis of pregnancy in very early gestation. Any pregnancy loss, which has not been confirmed ultrasonically or histologically, should be classified as a non-visualized pregnancy loss, irrespective of the time passed since the last menstrual period or clinical presentation. If the pregnancy has been diagnosed only by either serum or urine b-hCG, and the serial results decrease to negative, the pregnancy loss should be termed a biochemical pregnancy loss.

In the latest ASRM Practice Committee Opinion on the definition of recurrent pregnancy loss, a pregnancy is defined 'as a clinical pregnancy documented by ultrasonography or histopathologic examination' (ASRM Practice Committee, 2013).[1] They suggest a clinical evaluation should proceed following two first-trimester pregnancy losses, and, ideally, the threshold of three or more losses should be used for epidemiological studies.

As the diagnosis of RPL is based on self-reported losses which occurred in the past, it may not be accurate, as the women in the general population do not have their βhCG routinely measured and, consequently, their biochemical loss rate is underestimated. In contrast, women with RPL often have closer biochemical monitoring, which is less likely to be missed. The proportion of women with unexplained RPL (approximately one of three) may have environmental risk factors or endogenous pathologies not detected by current routine investigations.

In 1930s and 1940s, Malpas and Eastman suggested that the proportion to pregnancy losses in next pregnancy after three consecutive losses was as high as 73 -84%.[7] Years later, it has been demonstrated in several clinical studies that the risk of miscarriage is lower than what has been predicted.

Clinically recognised pregnancy loss is common occuring in approximately15-25% of pregnancies. Majority of sporadic losses before 10 weeks of gestation results from random numeric chromosome errors, specifically, trisomy, monosomy and polypoidy. In contrast, Recurrent Pregnancy loss is a distinct disorder defined by two or more failed clinical pregnancies. It is estimated that fewer than 5 % of women will experience two consecutive pregnancy miscarriages and only 1% experience 3 or more (ASRM practice committee, 2013).[1]

In the recent era, investigation and treatment is considered in couples with two consecutive spontaneous miscarriages, documented by ultrasound or histopathological examination (Leon Speroff et al).[8]

Evaluation is indicated when any of the following are present

1. Embryonic heart activity observed before any earlier pregnancy loss
2. Normal karyotype of the products of conception obtained from an earlier loss
3. Female partner over 35 years of age
4. Women with previous history of infertility

The risk of recurrent pregnancy loss in young women:

Table 1: women who have had atleast one live born infant[9]

Number of Prior miscarriages	% risk of miscarriages in next pregnancies
0	12%
1	24%
2	26%
3	32%
4	26%
5	53%

Future pregnancy outcome is significantly affected by previous reproductive history. Risk of miscarriage is found to be reduced in women who had prior live births when compared to women whose prior pregnancy has ended in miscarriage.

Table 2: Women who have not had atleast one live born infant[10]

Number of prior miscarriages	% risk of miscarriage in next pregnancies
2	24%
3	30%
4	40-50%

The prognosis for successful pregnancy depends both on the underlying cause and the number of previous losses. The chance of a viable birth even after four pregnancy losses is as high as 60%.

Prognosis for a viable birth After

One spontaneous loss :76%

Two spontaneous loss :70%

Three spontaneous loss: 65%

Four spontaneous loss: 60%

When a causative factor is identified, specific treatment can help in improving the prognosis for a successful pregnancy. The prognosis for a successful pregnancy outcome can be enhanced by determing the etiology, early identification of complications and being cautious of adverse maternal and perinatal outcomes.

REVIEW OF LITERATURE

Recurrent Pregnancy loss, affecting 1-2% of women of reproductive age seeking pregnancy, has been a clinical quagmire and a formidable challenge for the treating physician. There are many areas of controversy in the definition, aetiology, investigations and treatment of RPL. There is no identifiable cause in about 40-60% of these patients, in which case the condition is classified as idiopathic or unexplained RPL. The RPL investigations are extensive and should be undertaken in dedicated, specialized, well-equipped clinics/centres where services are provided by trained specialists. The challenges faced by the treating physician are even more overwhelming regarding the decision of what should be the most appropriate therapy offered to patients with RPL.

Patients presenting with RPL have experienced psychological trauma as they face the uncertainty of the outcome of the next pregnancy, and a variety of psychological and psychiatric disorders including anxiety, depression, posttraumatic stress disorders, and obsessive-compulsive disorders develop in these patients after RPL.[11]

EPIDEMIOLOGY OF PREGNANCY LOSS

Spontaneous pregnancy loss can be physically and emotionally taxing for couples, especially when faced with recurrent losses. It is a surprisingly common occurrence. Whereas approximately 15% of all clinically recognized pregnancies result in spontaneous loss, there are many more pregnancies that fail prior to being clinically recognized. Only 30% of all conceptions result in a live birth (Macklon NS et al, 2002).[12] Based on the incidence of sporadic pregnancy loss, the incidence of recurrent pregnancy loss should be approximately 1 in 300 pregnancies.[13] However, epidemiologic studies have revealed that 1% to 2% of women experience recurrent pregnancy loss (Stephensen MD , 1996).[14]

Although no reliable published data have estimated the probability of finding an etiology for RPL in a population with 2 versus 3 or more miscarriages, the best available data suggest that the risk of miscarriage in subsequent pregnancies is 30% after 2 losses, compared with 33% after 3 losses among patients without a history of a live birth (ACOG practice bulletin no.24).[15]

Nearly all chromosomally abnormal conceptions get aborted spontaneously before 10 weeks of gestation, and 90% of those with a normal karyotype continue.[16] Spontaneous abortion can also be viewed as a natural selection process for quality control by deleting chromosomally abnormal fetuses. Nearly about 30 - 60% of all conceptions will spontaneously get aborted within first 12 weeks of gestation; out of which half of the losses go unnoticed and this loss occurs even before a first missed menses.

AGE AND MISCARRIAGE

In recent decades, an increase in mean maternal age at childbirth in most high-resourced countries has been observed evidently. Advanced maternal age which is considered above 35years is being strongly associated with several adverse maternal and perinatal outcomes.[17] Advanced maternal age is considered as a major influencing factor for RPL due to the formation of chromosomally abnormal conceptions.[18] As age advances, there is increase in the incidence of mitotic segregation errors, rapid increase on number of aneuploid oocyte. Women with evidence of low ovarian reserve have an exceptionally higher rate of miscarriage, regardless of age.[19] Hence advancing maternal age adds up to the risk of

miscarriage in women with history of previous pregnancy losses. If both occult and recognized losses are being considered, total pregnancy loss in women above 40 years of age may exceed 75%.

Clifford K et al (1997), showed a 25% rate of miscarriages in women less than 30 years, increasing to 52% in a group of women older than 40 years.[20] They also concluded that increasing maternal age and number of previous miscarriages both had a negative effect on pregnancy outcome. To the contrary, a history of a live birth did not influence the outcome of the next pregnancy.

GESTATIONAL AGE AT THE TIME OF MISCARRIAGE

In a population of women with RPL, losses are likely to occur again at gestational ages similar to those previously documented.[21] These observations suggest the possibility of specific, but yet undiscovered, causes of loss that influence the viability of pregnancy at precise gestational ages. The chromosomal abnormalities are present in more than 90% of pre embrionic-aborted tissues, compared with only 6 to 12% of losses after 20 weeks gestation. The women with mid-trimester pregnancy loss represent a heterogeneous group with widely varying presentations

and origins. Fetal loss may have more than one cause, and the presence of dual or even triple pathologies increases the risk of a further late-term miscarriage or preterm delivery.[22] Mid-trimester pregnancy loss can be attributed to Antiphospholipid Syndrome and anatomic cervical incompetence.

PROGNOSTIC VALUE OF TRANSVAGINAL ULTRASOUND OBSERVATIONS

The risk of miscarriage decreases as the duration of pregnancy advances. The risk of pregnancy loss falls progressively after appearance of gestational sac (12%), yolk sac (8%), as crown rump length increases, (greater than 5mm – 7%, 6 – 10mm – 3%, more than 10 mm – less than 1%).The important ultrasound findings in pregnancies that subsequently resulted in fetal loss, compared to those resulting in live births, were a small gestational sac diameter and fetal bradycardia.

Appearance of embryonic cardiac activity by approximately 6 weeks is another important developmental mile stone and good prognostic indicator. In both normal and infertile asymptomatic young women, the

appearance of embryonic heart activity decreases with the risk of pregnancy losses from 12% - 15% to 3 – 5%.[23]

ETIOLOGY OF RECURRENT PREGNANCY LOSS

1) Genetic factors : 3.5 – 5 %

2) Anatomical factors : 12 -16 %

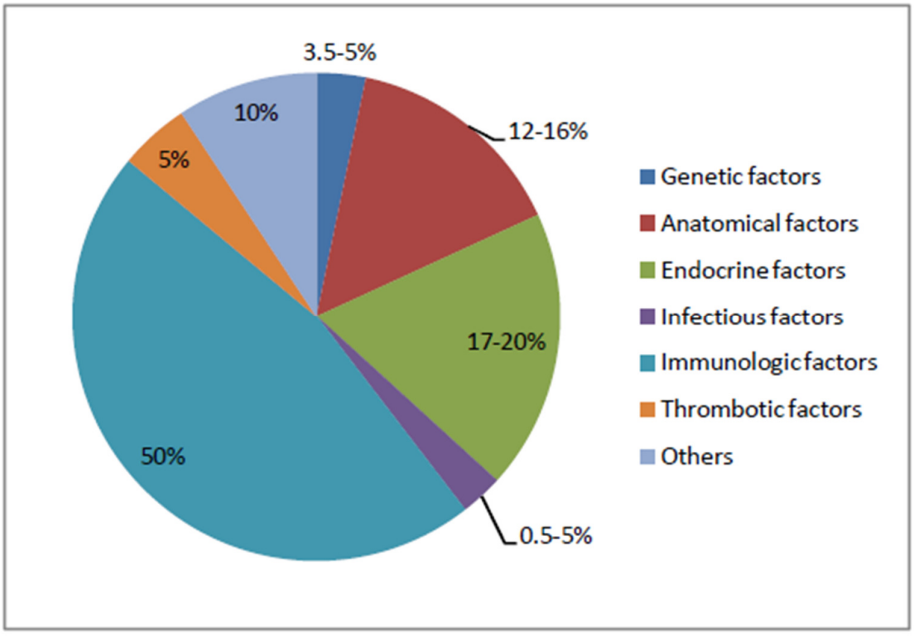
3) Endocrine factors : 17 – 20 %

4) Infectious factors : 0.5 – 5 %

5) Immunologic factors : 20 -50 %

6) Thrombotic factors

7) Others: 10 % (environmental, placental abnormalities, male factors, medical illness, altered uterine receptivity, exercise)



GENETIC FACTORS

A considerable proportion of pregnancy losses are due to genetic abnormalities in the embryo. Many of them are due to de novo non disjunctional events but balanced parentral translocations are responsible for small yet significant proportion of genetic abnormalities in couples with recurrent pregnancy loss. In recent era, molecular genetic technology provide ample amount of genetic information about other genetic causes and risk factors for recurrent pregnancy loss.

Large number of abortuses so far cultured and karyotyped suggest that approximately 50 % of all first trimester pregnancy losses, 30% of second trimester abortuses, and 3 % of still births are chromosomaly abnormal.[24]

Chromosomal abnormalities:

90% - numerical (aneuploidy, polyploidy)

10% - structural abnormality (translocation , inversion and mosaicism)

Numerical chromosomal abnormalities:

The remarkable inefficiency of human reproduction is largely the result of spontaneous fetal aneuploidy. Nearly 50 – 70 % of sporadic spontaneous

abortions have some form of cytogenetic abnormality, the most common abnormalities being autosomal trisomies (60%), monosomy X (20%) and polypoidy (20%) (Silver and branch, 2007).[25] Most of these defects are due to random errors in germ cell development which equally affects pregnancies regardless of history of RPL.

Typically, numerical aneuploidy results from meiotic non disjunction in the germ cells in couples with normal karyotypes, and the recurrence of a particular abnormality in future pregnancies is rare in patients with RPL and in the general population (Warren and silver, 2008 ; suzumori and Sugiura-Ogasawara, 2010).[26,27] So most of the losses in RPL are the result of random, non recurring events and hence the prognosis for subsequent of pregnancies in RPL couples is better after an aneuploid miscarriage than after an euploid miscarriage (Warburton et al, 1987; Ogasawara et al 2000; Carp et al , 2001).[28,29,30]

Autosomal trisomy are the most common usually involving chromosome 13 – 16, 21 or 22, followed by monosomy X (45 X),42,44,45,21. The likelihood of euploid abortus increases with the number of previous miscarriages and after a previous abortion having a normal karyotype.

Structural chromosomal abnormlities:

Structural chromosomal abnormalities are an unequivocal explanation for repetitive abortions. The most common structural rearrangement encountered is a translocation, found in about 5% of couples experiencing repeated losses. Individuals with balanced translocations are phenotypically normal, but their offspring (abortuses or abnormal live-born infants) may show chromosomal duplications or deficiencies as a result of normal meiotic segregation. Among couples with repetitive abortions, about 60% of translocations are reciprocal and 40% robertsonian. Females are about twice as likely as males to show a balanced translocation.

Inversions

Inversions are uncommon parental chromosomal rearrangements, but responsible for repetitive pregnancy losses analogous to translocations. In inversions, the order of genes is reversed. Individuals heterozygous for an inversion should be normal if their genes are truly just rearranged. However, individuals with inversions suffer untoward reproductive consequences as result of normal meiotic phenomena. Pericentric inversions are present in perhaps 0.1% of women and 0.1% of men

experiencing repeated spontaneous abortions. Paracentric inversions are even rarer.

In the evaluation of RPL, Parents should undergo peripheral karyotyping in order to detect any balanced structural chromosomal abnormalities. Balanced reciprocal translocations and robertsonian translocations are observed in about 2-5% of couples with recurrent miscarriage. Genetic counselling is important when a structural genetic abnormality is detected. The likelihood of subsequent healthy live birth depends on the chromosome(s) involved and the type of rearrangement. When one of the partners has a structural genetic abnormality, preimplantation genetic testing (PGT), amniocentesis, or chorionic villus sampling are options to detect the genetic abnormality in the offspring. Treatment options include preimplantation genetic diagnosis (PGD) for specific translocations , with transfer of the unaffected embryos, or the use of donor gametes.

Karyotyping data are likely to underestimate the prevalence of chromosomal abnormalities due to unrecognized maternal cell contamination; normal euploid cells are less likely to fail culture than abnormal cell lines.[31] Some genetic abnormality cannot be detected using standard cytogenetiic techniques like isolated gonadal / germ line mosaicism/ single gene defects.

Analysis using newer techniques not dependent on cell culture (FISH – fluorescence in situ hybridization, CGH – comparative genomic hybridization) suggest that the true incidence of chromosomal abnormalities in miscarried early pregnancies is closer to 75%.

ANATOMIC FACTORS

CONGENITAL UTERINE MALFORMATIONS

Congenital anomalies are found in 8.4%–12.6% of women with RPL, which is seven to eight times higher than the general population.(Jaslow, 2014; Grimbizis GF, 2001).[32,33]

Congenital abnormalities are the consequence of an abnormal development of the Müllerian ducts and include septate, bicornuate, unicornuate, didelphic, and arcuate uteri. They are reportedly found in up to 10% of women with RPL (Jaslow CR, 2014).[32]

Septate uterus is the most common uterine developmental anomaly accounting for 80-90% in both women with recurrent miscarriage (3.5% prevalence) and in general population. Miscarriage rate associated with septate uteri is 65 %.

Hysteroscopic septoplasty dramatically improved post operative pregnancy outcomes. (80% term, 5% preterm, 15% miscarriage). Arcuate uteri are a normal variant. Residual septal defect less than 1cm has no adverse effect on pregnancy.

A septate uterus leads to poor reproductive outcome:

1.Poor septum vascularization leading to poor decidualization and placentation (Dabirashrafi 1995, Kupesic 1998, Kupesic 2001).[34,35,36]

2. Increased amount of muscle tissue in the septum can cause miscarriage by uncoordinated contractility

3.Reduced length of unaffected uterine cavity

4.Local defect of VEGF receptors (Raga Fertil Steril 2009).[37]

Canalization defects appear to reduce the chance of clinical pregnancy and increase the chance of miscarriage and preterm delivery. These are more profound in cases of septate uteri. Unification defects do not reduce fertility but some defects, in particular bicornuate uteri, are associated with aberrant outcomes throughout the course of pregnancy. Cases of arcuate uteri, often considered an incidental benign finding, are specifically associated with poor outcomes in late pregnancy, i.e. second-trimester miscarriage.

There is a lack of randomized controlled trials (RCTs) concerning the impact of uterine metroplasty on reproductive outcomes in women with congenital uterine anomalies and RPL. Septate uterus is the most common type and has more chance of spontaneous abortion(Grimbizis GF et al, 2001).[33] There is evidence to suggest improved pregnancy rates following metroplasty (Valle RF et al ,2013) and it is highly recommended to do metroplasty in women with RPL having septate uterus.[38]

The prevalence of arcuate uteri is the same as in general population, and their effect on reproductive outcome remains controversial (Grimbizis GF, 2001).[33] Therefore, arcuate metroplasty is not recommended in women with RPL (practice committee ASRM, 2012; Jaslow ; Valle).[1,32,38] The other congenital abnormalities are more commonly associated with third trimester pregnancy loss and preterm birth, and the decision to treat or not is more complex (Grimbizis GF).[33]

Metroplasty is not recommended for unicornuate uteri, is highly controversial for didelphys, and is only recommended as a last resort for bicornuate uteri (Alborzi S et al, 2009; Brucker SY, 2011).[39,40] Finally, it should be noted that for women with RPL secondary to irreversible uterine anatomic defects, the use of a gestational carrier is a viable option.

DES EXPOSURE

Uterine anomaly was found in almost 70% of women exposed to DES which is rare nowadays . The common uterine abnormality is T shaped uterus, hypoplastic uterus, irregular intrauterine filling, constriction rings. As the women who have been exposed are beyond their reproductive age, women encountered with such abnormality is rare at present. The risk of spontaneous abortion is 24% and risk of ectopic pregnancy is increased by 9%.

UTERINE LEIOMYOMA

Some research has linked the presence of uterine fibroids with recurrent miscarriage in women, but the data thus far had been inconclusive. They impede embryonic implantation and causes poor regional blood flow. The expression of *HOX10*, a gene that controls differentiation and is involved in implantation, is found to be low in uterus with fibroids.

Hysteroscopic Myomectomy should be considered in cases of submucosal fibroids or any type fibroids larger than 5 cm. Resection has been shown to significantly improve live birth rates from 57% to 93%. Saravelos et al,2011 found that women with fibroids distorting the

uterine cavity had a total miscarriage rate of 76.7 percent and a live birth rate of 23.3 percent.[41] Women with cavity-distoring fibroids also had a higher mid-trimester miscarriage rate (21.7 percent) than the group of women with unexplained recurrent miscarriages (8 percent). They found that women who had cavity-distorting fibroids and underwent hysteroscopic myomectomy had significantly improved outcomes, with a significant drop in the mid-trimester miscarriage rate and a live birth rate increasing from 23.3 % to 52 %.

INTRAUTERINE ADHESIONS

Decreased functional intrauterine volume, endometrial fibrosis and inflammation predispose to placental insufficiency. Pregnancy outcomes are generally poor. 40 – 80 % results in spontaneous miscarriage and 25 % ends in preterm delivery. The prognosis generally correlates with the severity of the disease. Hysteroscopic adhesiolysis dramatically improves the pregnancy outcome.

CERVICAL INCOMPETENCE

The diagnosis is based on a history of late second trimester miscarriage

characterized by painless cervical dilatation followed by ballooning of membranes and ultimately resulting in expulsion of fetus. Efficient treatment helps in preventing future miscarriage.

IMMUNOLOGICAL PHENOMENON

Pregnancy is a immunotolerance state. A subset of CD4 cells describe CD25 on their cell surface. These CD4, CD25 cells are called Regulatory T Lymphocytes (T reg cells). T reg cells when activated by autoantigens can suppress activated inflammatory cells.

Cellular Immune Mechanisms

Decidual NK cells comprises 70-80% of the total endometrial population at the implantation site. Human reproductive tract is populated by TCR γδ+ they are increased in early pregnancy; their functions are direct, non – MHC registered recognition of antigens within tissues. Suppressor macrophages helps in pregnancy maintenance. They help in promotion of anti – inflammatory effect. T reg cells suppress maternal responses to self and to the fetus.

Antigen Presentation at the Maternal Fetal Interface

Implanting trophoblastic allograft causes down regulation of its expression of the MHC – encoded transplantation antigens and avoid recognition as non – self. Current theory, in placental trophoblast MHC class I and II molecules are not expressed.[42]

Extravillous trophoblast express classical MHC class I HLA – C and non – classical HLA – E and G products. They are characterized by their invasive potential deep into the maternal decidua, an activity essential for proper placental development. Aberrant expression of class II MHC; over expression of class I MHC leads to cytotoxic Tcell attack, could enhance abortion. MHC II genotypes appear to affect susceptibility to a variety of diseases, like diabetes and other auto immune diseases and adverse pregnancy outcome.

Regulation of maternal decidual cells

Regulatory mechanisms include

i) Alterations in T- helper cell phenotypes

ii) Reproductive hormones and immunosuppression

iii) Tryptophan metabolism

CDT4 cells are divided into TH1 responses and TH2 responses. TH1 cells are associated with inflammation. They are harmful to the implanting embryo. TH2 cells are associated with IL – 4,5,6,10 and antibody production; they are important for pregnancy maintenance.[43] The dysregulation of their T-helper cellular immune response accounts for about 60-80%. Leukemia inhibitory factor is absolutely essential for pregnancy maintenance.

Humoral Immune Response

Patients with recurrent pregnancy loss display altered humoral responses to endometrial and trophoblast antigens; Historically these IgG and IgM antibodies (cardiolipin and phosphatidylserine) are thought to be directed against negatively charged phospholipids. More recently the high titre of antibodies directed against a protein cofactor, called β2 glycoprotein I alone is sufficient for diagnosis.

LABORATORY ASSESSMENT (SAPPORO CRITERIA).[44]

Antiphospholipid antibody syndrome requires presence of atleast 1 clinical and 1 laboratory Criterion

• Clinical

1. One or more confirmed episodes of vascular thrombosis (venous, arterial, or small vessel).

2. Pregnancy morbidity

Pregnancy complications including

- ✓ 1 or more fetal deaths at greater than 10 weeks of gestation of a morphologically normal fetus.
- ✓ One or more premature birth of a normal neonate before 34th week because of pre eclampsia, or placental insufficiency .
- ✓ 3 or more consecutive pregnancy losses at less than 10 weeks of gestation.

• Laboratory (repeated at least 2 times, more than 12 weeks apart)

- ✓ Positive plasma levels of the anticardiolipin antibodies (IgG Or IgM) at medium to high levels
- ✓ Positive plasma levels of the lupus anticoagulant
- ✓ Anti β2 – glycoprotein 1 antibody of IgG or IgM isotype in 99th percentile titre.

The incidence of APLAs in patients with RPL is between 3-5%; Less favourable pregnancy outcome was noted among patients with known

Systemic Lupus Erythematosus. Anti-thrombotic molecule (annexin V) is reduced within the placental villi resulting in atherosclerosis in decidual spiral arteries.

THROMBOPHILIAS

Abnormal placental vascularisation and inappropriate placental thrombosis would relate these thrombophilic states to pregnancy loss. The incidence of inherited thrombophilic mutations in Caucasians – 15%;

The most common are

I) Factor V leiden – 5%

II) Prothrombin promoter region mutation – 2-3%

III) Mutation in gene MTHFR – 11-15% .They are associated with mild thrombotic risks.

Thrombophilic gene defects

The genetic defects involved in the ever-expanding group of inherited thrombophilias are perhaps the best studied single gene mutations with reference to RPL. Among these, the majority of reports have addressed factor V Leiden, prothrombin gene promoter mutations, activated protein C resistance, and mutations in methylenetetrahydrofolate reductase,

plasminogen activator inhibitor, thrombomodulin, and annexin A5 genes. Unlike trinucleotide expansion disorders and immune–related single gene mutations, the gene mutations causing several of the inherited thrombophilias are seen in relatively high prevalence in select populations. The data linking these defects to RPL, however, are conflicting. Mutations in factor V Leiden, the most common genetic cause of thrombosis, have a twofold higher prevalence in women experiencing repeated miscarriages compared with controls.[45,46,47,48,49,50] Mutations in the gene encoding Annexin A5, a protein that acts as an anticoagulant in placenta villi, have also been associated with a twofold increase in RPL risk.[51] Other cohort studies, however, have failed to confirm an association between RPL and inherited thrombophilias such as factor V Leiden and prothrombin promoter gene mutations and carriers and noncarriers of annexin A5 mutations have similar live birth rates, limiting the clinical significance of this particular mutation.[45,52] In fact, there are data showing that when stratified for gestational age at the time of fetal demise, maternal carriage of the FVL mutation protects against pregnancy loss occurring before 10 wk of gestation.[53] This finding aligns with earlier data showing increased implantation rates in carriers of FVL who become pregnant through in vitro fertilization.[54] Together these studies support the concept

that FVL increases implantation of compromised embryos that would not typically implant and are ultimately lost.

Despite the growing evidence that thrombophilias caused by single gene defects may be associated with adverse pregnancy outcomes, the absolute risk of adverse outcomes remains low. Thus, current recommendations do not support universal screening for inherited thrombophilias in women with a history of pregnancy loss.[55,56,57] Both the specific thrombophilia and the type of pregnancy loss (isolated vs. recurrent; early vs. late) contribute to the vast spectrum of documented associations and the underlying pathophysiologic mechanisms used to support these associations.[58]

Despite the present recommendations, however, many practitioners will screen for prevalent inherited thrombophilias (factor V Leiden, prothrombin gene mutations and methylenetetrahydrofolate reductase in Caucasian patients; protein C, protein S and antithrombin deficiencies in some patients of eastern Asian descent) in RPL patients who have a first-degree relative with a known or suspected thrombophilia or who report a personal history of venous thromboembolism.[1]

That said, data supporting treatment of a diagnosed gene defect associated with an inherited thrombophilia in the absence of a personal thrombotic history are weak. Although observational studies have shown a decrease in pregnancy complications in women with the Factor V Leiden mutation or mutation in the prothrombin promoter region that are prophylactically treated with low molecular weight heparin, there is a lack of randomized control studies that confirm these same findings.[59]

Recently, two randomized controlled trials investigated the empiric use of aspirin alone or a combination of aspirin with low molecular weight heparin in women with RPL and found no improvement in live birth rate when compared with placebo.[60,61] Based on current evidence, therefore, the empiric use of antithrombotic agents in women with unexplained RPL is not recommended.[4,61]

More randomized placebo-control trials are needed to show both a mechanistic association between inherited thrombophilias and RPL and successful treatment options to improve live birth rates in couples suffering from recurrent miscarriage.

The current studies on maternal-fetal interface immunity revealed that both second-trimester abortion and spontaneous preterm birth, similar to those of Preeclampsia and Fetal growth restriction, suffered from abnormal immune cell function and cell number, leading to disorders of maternal-fetal tolerance and uterine spiral artery reconstructions (A.Ekin et al, 2015).[62] Recent reports have shown that about 50-65% of women with unexplained spontaneous abortion have at least one inherited or acquired prethrombotic state (PTS) [G.Mitic et al, 2011].[63] In PTS, FVL mutation raises the risk of early miscarriages and early intrauterine fetal deaths [M.A.Rodger et al,2011].[64] Meta-analysis has shown that late spontaneous abortion is closely related to factors V and II (prothrombin) mutations and to the lack of protein S induced congenital thrombosis [G.Kovalevsky et al ,2004].[65]

Clinically, patients with RSA are not recommended for routine hereditary thrombosis test, in that mutations of factors V and II (prothrombin) in domestic Han population are rare. Therefore, tests of FVL, coagulation factor VIII, antithrombin, and protein C are only recommended for patients with a thrombosis family history and who suffered from unexplained FRSA.

INFECTIOUS CAUSES

Spontaneous miscarriage has found to be associated with Chlamydia trachomatis, Ureaplasma urealyticum or Mycoplasma hominis infection. Toxoplasma gondii, Listeria monocytogenes, Campylobacter species, herpes virus, and cytomegalovirus has also been implicated. Many studies has been conducted to correlate the association between miscarriage risk and Bacterial vaginosis. There is 5 fold increased risk of pregnancy loss before 20 weeks of gestation when Bacterial Vaginosis was diagnosed at the first prenatal visit before 14weeks.[66]

Chronic subclinical endometritis in women with symptomatic lower genital tract infection explains association between infection and miscarriage. Chronic endometritis (CE) is defined as chronic inflammation of the endometrial lining, and some studies have shown an increased prevalence in women with RPL (10%–27%).[67,68,69] Endometrial receptivity is thought to be impaired by the stromal infiltration of plasma cells, as well as altered expressions of genes involved in implantation, leading to RPL but also infertility and recurrent implantation failure following in vitro fertilization (IVF).[69]

Several methods have been used to confirm the diagnosis of CE, but the gold standard is identification of plasma cells in the endometrial stroma, using immunohistochemistry stains for syndecan-1 (CD138), a marker of plasma cells.[69] The etiology is most likely infectious, and many treatment regimens with antibiotics have been proposed, the most widely used being doxycycline (200 mg per day for 14 days), with some studies reporting favorable outcomes following treatment.[67,68,69] However, no randomized trials have been published to date, and controversies remain concerning the impact of CE on reproductive outcome, the patient population to screen, the treatment regimen, and need for a biopsy to confirm resolution.

Given the lack of conclusive evidence, several international societies do not include screening for CE in their recommendations.[1,4] Several other infections have been investigated as potential causes of early miscarriage. There is some evidence that bacterial vaginosis (Mycoplasma hominis, Ureaplasma urealyticum), brucellosis, syphilis, cytomegalovirus, dengue fever, human immunodeficiency virus, rubella, and malaria are more frequently found in women with spontaneous miscarriage.[70] However, no causal link has been established, and it is not recommended to test or empirically treat asymptomatic women with RPL.

ENVIRONMENTAL FACTORS

SMOKING: Smokers who consume 10 cigarettes / day has adverse pregnancy outcome. The anti metabolite composition of cigarette like nicotine, carbondioxide and cyanide may predispose to placental insufficiency .

ALCOHOL: Consumption exceeding two drinks / day increases the risk. It adds to the additive risk with smoking.

CAFFEINE: Heavy caffeine consumption more than 300 mg / day, equivalent to 3 cups / day - 2 fold increased risk of spontaneous miscarriage.

OBESITY: A body mass index equal to or greater than 25 is associated with greater risk of miscarriage.[71]

Others

Anaesthetic gases (Perchorethylene)

- ✓ Exposure to heavy metals (Mercury,Lead)
- ✓ Isotretinoin (Accutane)
- ✓ Painters and factory workers.

Male factors

Sperm samples from recurrent pregnancy loss couples have an increase in their sperm DNA fragmentation.[72,73,74] Meta-analysis showed a significant increase in miscarriage in patients with high DNA damage compared with those with low DNA damage.[75] The associating sperm quality with recurrent pregnancy loss emphasizes the importance of evaluating male factor by tests. Several different tests are available, but no consensus has yet been reached as to which tests are most predictive.

Among terminal uridine nick-end labeling assay (TUNEL), sperm chromatin structure assay (SCSA), sperm chromatin dispersion (SCD), and alkaline Comet assays, the alkaline COMET assay showed better prediction for male infertility.[76] A chromosomal abnormality was found in 15.2% men with azoospermia and in 2.3% nonazoospermic men.

Male factors abnormality is a significant cause for recurrent pregnancy loss after assisted conception. The number of azoospermic men who needs to be screened to prevent one miscarriage is 80–88 and the number need to screen is 315–347 in the nonazoospermic group.[77] Although there is some evidence of association between DNA defragmentation and

recurrent miscarriage, well-designed prospective studies are needed before using these tests in clinical practice.[78] Routine testing for spermploidy (e.g. fluorescence *in situ* hybridization [FISH]) or DNA fragmentation is not recommended.

UNEXPLAINED RECURRENT PREGNANCY LOSS

Even after thorough evaluation, half of the women with recurrent miscarriage have no identified predisposing factors. Frequent communication, cautious optimism and emotional support during the first trimester of the next pregnancy have their own therapeutic value. 75 % of women ultimately achieve successful pregnancy. Empiric
treatment with exogenous progesterone or aspirin in women with unexplained recurrent pregnancy loss has no proven value.

ENDOCRINE FACTORS

LUTEAL PHASE DEFECT

Early pregnancy depends upon the secretion of progesterone from corpus luteum till 7 weeks of gestational period. Corpus luteal shift do not occur suddenly, it occurs gradually over a period of 7-9 weeks of pregnancy.[79]

The human implantation window is relatively narrow approximately 6-10 days after ovulation. Low levels of circulating progesterone causes delayed endometrial maturation and causes shift in implantation window leading to failed or late implantation. Luteal phase defect is diagnosed when there is a persistent delay of longer than 2 days in the histologic development of the endometrium compared with the respective day of the menstruation.

A low progesterone concentration during early pregnancy reflects defective corpus luteum, an intrinsically abnormal conceptus or both. Serum progesterone concentration fluctuates throughout the day as the corpus luteum progesterone secretion is pulsatile. Measurement of serum progesterone in early pregnancy to assist the quality of luteal function and supporting the risk pregnancy with exogenous progesterone therapy are futile.

Some of the cases of luteal phase defect are associated with hypersecretion of luteinizing hormone (LH). Abnormal LH causes premature aging of oocyte, dyssynchronous maturation of endometrium, improperly timed endometrium at potential implantation sites.[80] The Luteal phase deficiency

is considered as a subtle form of ovulation dysfunction . Some prefer to treat with exogenous progesterone supplementation starting from 2-3 days after ovulation.[81]

POLYCYSTIC OVARIAN DISEASE

Polycystic ovarian disease are not a characteristic feature of a specific endocrine disorder, results from functional derangement in follicular development, or by sustained increased intraovarian androgen levels as a consequence of chronic anovulation.

The National Institute of Child Health and Human Development (NICHD) in 1990.

1. Hyperandrogenism
2. Menstrual dysfunction
3. Exclusion of other disorders having a same clinical presentation .

The European society for Human Reproduction And Embryology (ESHRE) and The American Society Of Reproductive Medicine (ASRM) – Rotterdam criteria -2003, at least two of three major criteria including,

1. Oligo/anovulation
2. Clinical or biochemical signs of hyperandrogenism
3. Polycystic ovaries as identified by ultrasound (total number of follicles, 12 or more measuring 2 – 9 mm in diameter ; ovarian volume > 7 – 7.5 ml)

The overall prevalence of insulin resistance among with PCOS is 50 -75%, greater in obese than in lean individual. Metformin treatment can reduce or eliminate the higher risk of miscarriage in women with PCOS relating to an underlying metabolic disorder.

THYROID HORMONES IN PREGNANCY

Moderate enlargement of thyroid gland by glandular hyperplasia and increased vascularity occurs during pregnancy, but normal pregnancy does not typically cause thyromegaly. In early first trimester, oestrogen increases the synthesis of thyroxine binding globulin. Serum levels of total thyroxine T4 increases beginning from 7 and 9 weeks, and plateau at 18 weeks. Rise in Free serum T4 levels peaks with hCG levels, later returns to baseline . T3 levels starts increasing and reaches peak at 18 weeks later plateaus. TRH levels are not increased during normal pregnancy. TSH

levels get supressed during pregnancy. This results in failure to diagnose women with early hypothyroidism. Inverse relationship exists between TSH and hCG . TSH levels drops to a nadir at 10 weeks and at the same time hCG reaches the peak. As hCG decreases later, TSH raises in later part of pregnancy. Fetal thyroid gland starts secreting thyroxine only after 10 – 12 weeks of pregnancy, since then the entire supply and fetal brain development depends on maternal source.

Hypothyroidism in pregnancy

Hypothyroidism, even subclinical have increased risk of spontaneous miscarriage. Mechanism being impaired function of endometrium, corpus luteum and the placenta. Preconceptional screening with achievement of euthyroid state before pregnancy is necessary for successful outcome.

Universal screening is not routinely recommended. But those with elevated TSH levels needs estimation of anti thyroid antibodies. Women with positive thyroid antibodies have has an increased risk of becoming hypothyroid as pregnancy advances. Women treated for hypothyroidism requires an increase in thyroxine during pregnancy starting as early as 5th week of gestation.

The causes for such an effect are

1. estrogen induced increase of thyroid binding globulin,

2. dilutional effect of increase in vascular volume ,

3. and the increase in placental transport and metabolism.

As soon as pregnancy is diagnosed, 30 % increase in levothyroxine dose is required . TSH levels should be monitored monthly and adjust levels in lower limit of normal range for trimester specific levels .

Recurrent pregnancy loss: current perspectives

Over the years, evidence-based treatments such as surgical correction of uterine anomalies or aspirin and heparin for antiphospholipid syndrome have improved the outcomes for couples with recurrent pregnancy loss. However, almost half of the cases remain unexplained and are empirically treated using progesterone supplementation, anticoagulation, and/or immunomodulatory treatments. Regardless of the cause, the long-term prognosis of couples with recurrent pregnancy loss is good, and most eventually achieve a healthy live birth. However, multiple pregnancy losses can have a significant psychological toll on affected couples, and

many efforts are being made to improve treatments and decrease the time needed to achieve a successful pregnancy.

In parallel, epidemiological evidence has shown that the prospects after recurrent miscarriages can be excellent. Moreover, factors derived from the obstetric history, e.g., maternal age and number of preceding miscarriages, independently have a negative impact on the prognosis. Miscarriages have also been linked to subfertility.

Agrawal S et al, (2015) In this study, author studied the pregnancy outcome in patients with history of previous spontaneous abortions.[82] It is prospective study that included a total of 70 patients with history of previous spontaneous abortion and the patients were observed for complications. The final outcomes were term live birth (74.3%), abortion (14.3%), preterm delivery (8.6%), and still birth (2.8%). Caesarian section was done in 23.3% patients for various indications. They concluded that thers is increased risk of abortion, preterm delivery, need for caesarean sections and fetal loss in cases of previous spontaneous abortions.

David.H.Thom et al had evaluated the association between spontaneous abortion and subsequent adverse birth outcomes.[83] They concluded that women with three and more spontaneous abortions were at higher risk of preterm < 37 weeks of gestation(**95%**), placenta previa (95%), premature membranes rupture > 24 hours, Breech presentation (95%), congenital malformation (95%).

Eva Alberman et al have compared birth weight of babies in patients with previous live birth and in patients with spontaneous abortion.[84] The important observations were that the mean birth weights of babies preceding a spontaneous fetal loss was lower than that of live births preceding another live births and that in the subgroup of women with repeated early losses, mean birth weight fell with increasing pregnancy order.

JoaquinE.Paz et al did a study to find out the association between previous fetal loss and fetal malformation and low birth weight in the subsequent pregnancies.[85] They found out that multiple malformation, Downs syndrome, anencephaly, Spina bifida, Talipus Equinovarus, Congenital dislocation of hip and low birth weight are associated with previous fetal

loss and they finally suggested that abortion or a still birth in a previous pregnancy should be taken into consideration when the risk of malformation or low birth weight in a subsequent pregnancy is assessed.

S.A.Brigham et al did a study to find out how many fetuses continued pregnancy and survived in women with idiopathic recurrent miscarriage.[86] They also showed that there is decrease in pregnancy success rate with increasing number of previous abortion.

P.W.Reniald, R.W.Beard et al did a study in which they have compared the outcomes of pregnancies progressing beyond 28 weeks of gestation in women with a history of recurrent miscarriage.[87] Out of the 97 women who had 3 miscarriages 30 percent were small for gestational age, 28 percent were born preterm and perinatal mortality was 161/1000 births, all the parameters are significantly increased above the prevalence for a normal obstetric population.

Eva Alberman, Eve Roman et al The most important observations were that the mean birth weight of babies preceding a spontaneous fetal loss was lower than that of live births preceding another livebirth, and that in

the subgroup of women with repeated early losses, mean birth weight fell with increasing pregnancy order.[88]

M.J.N.C.Keirse, R.W.Rush et al did a study about risk of pre-term delivery in patients with previous pre-term delivery and/or abortion.[89] Patients with a history of two or more abortion had an increased risk of spontaneous pre-term labour and delivery in future pregnancies. This increased risk related mainly to previous second trimester abortions and not to previous first trimester abortions. Patients with one previous spontaneous pre-term labour and delivery had a 37 per cent risk, and those with two or more pre-term deliveries a 70 per cent risk of again delivering pre-term. There appeared to be no beneficial effect of cervical suture on the incidence of pre-term delivery in these patients.

Ulla Breth Knudsen, Villy Hansen, did a study on the prognosis of a new pregnancy following previous spontaneous abortions.[90] The risk for a clinical spontaneous abortion in a pregnancy following 0 to 4 consecutive spontaneous abortions was estimated. The overall risk for spontaneous abortion was 11% and the risk for a spontaneous abortion was 16, 25, 45 and 54% after 1 to 4 previous consecutive spontaneous abortions, respectively. For women over 35 years, the risk for spontaneous

abortion was significantly increased, but the almost identical abortion rates after repeated abortions in both young and old women indicate a risk factor which is not age-related**.**

Eyal Sheiner, Amalia Levy et al did a study to examine the association between spontaneous consecutive recurrent abortions and pregnancy complications such as hypertensive disorders, abruptio placenta, intrauterine growth restriction and cesarean section (CS) in the subsequent pregnancy.[91] A population-based study comparing all singleton pregnancies in women with and without two or more consecutive recurrent abortions was conducted.. Results: Using a multivariate analysis, with backward elimination, the following complications were significantly associated with recurrent abortions—advanced maternal age, cervical incompetence, previous CS, diabetes mellitus, hypertensive disorders, placenta previa and abruptio placenta, mal-presentations and PROM. A higher rate of CS was found among patients with previous spontaneous consecutive recurrent abortions (15.9% versus 10.9%; $P < 0.001$). Another multivariate analysis was performed, with CS as the outcome variable, controlling for confounders such as placenta previa, abruptio placenta, diabetes mellitus, hypertensive disorders, previous CS, mal-presentations,

fertility treatments and PROM. A history of recurrent abortion was found as an independent risk factor for CS (95% ; P < 0.001) .They concluded that a significant association exists between consecutive recurrent abortions and pregnancy complications such as placental abruption, hypertensive disorders and CS. This association persists after controlling for variables considered to coexist with recurrent abortions. Careful surveillance is required in pregnancies following recurrent abortions, for early detection of possible complications

AIM AND OBJECTIVES

Aim

The aim of our study is to estimate the risk of the Preterm delivery, low birth weight, IUGR, recurrence of abortion, still birth, IUD, PROM or any other adverse outcome in women with history of recurrent pregnancy loss.

Objectives

1. To test the hypothesis that previous unfavourable pregnancy outcome increases the risk of adverse outcome in the present pregnancy.
2. To look for association between previous spontaneous abortion and preterm delivery, low birth weight, IUGR, recurrence of abortion, still birth, IUD, PROM in subsequent pregnancies.

MATERIALS AND METHODS

Sample size- 400 cases

This prospective study was carried out in the Department of obstetrics and gynecology, Thanjavur Medical college and hospital, Thanjavur.

Period of study-.June 2016 to June 2017

Inclusion criteria

1. Cases (200): patients with history of 2 or more spontaneous abortion at less than 20 weeks, irrespective of the period of gestation were included.

   Controls (200) :Each Primigravida who delivered or aborted subsequent to each women in case group.

2. Age group-18 to 35 years.

Exclusion criteria

1. Patients with induced abortion

2. History of spontaneous abortion with twin gestation

3. History of ectopic pregnancy.

OBSERVATION AND RESULTS

Study Design:

A Case –control clinical prospective study with 200 patients with history of recurrent pregnancy loss(Cases group) and 200 primigravidae who delivered subsequently to the cases (control group) were undertaken to compare the obstetric outcomes between the two groups.

Table 3: Age distribution of patients

AGE IN YEAR	CONTROL		CASE		COMBINED	
	NO	%	NO	%	NO	%
18-20	10	5	0	0	10	2.5
21-25	120	60	44	22	164	41
26-30	62	31	138	69	200	50
31-35	8	4	18	9	26	6.5
TOTAL	200	100	200	100	400	100
MEAN ± SD	24.02±3.30		27.84±3.36		25.93±3.33	

From table 3, the mean age of patients in case group is 27.84 and control group is 24.02. Indicating that most patients in case group were distributed predominantly in the 26-30 age group whereas patients in control group falls mostly under 21-25 age group.

Fig 1: Age distribution of patients

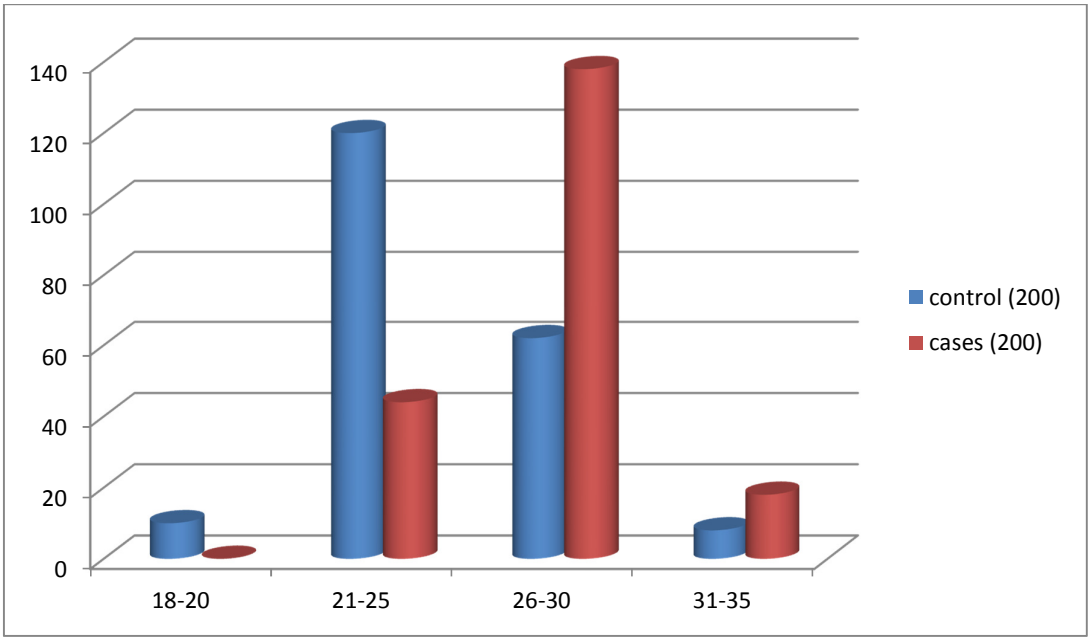


Table 4: Distribution of cases group with respect to the number of previous abortions.

PREVIOUS HISTORY OF ABORTIONS	NUMBER	PERCENTAGE
2	169	84.5
3	26	13
4	3	1.5
5 or more	2	1
TOTAL	200	100

Table 4 shows the distribution of cases with respect to the number of previous abortions**.** 84.5% of patients had 2 previous abortions, 13 percent of patients had previous 3 abortions, 1.5 percent of patients had 4 previous abortions and 1 percent of patient had previous 5 or more abortions.

Fig 2: Distribution of cases group with respect to the number o previous abortions

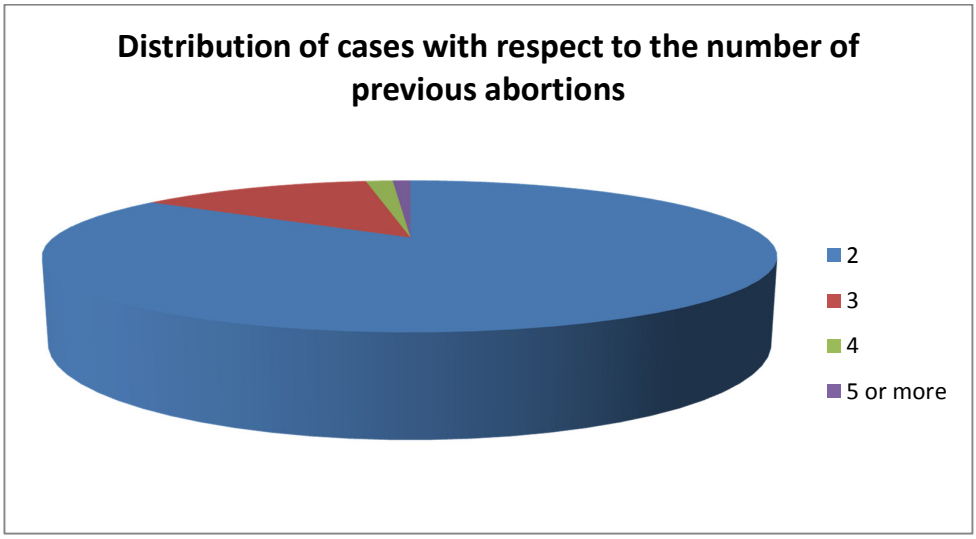


Table 5: Comparison of obstetric outcome between the cases and controls

S.NO	OUTCOME	CONTROL		CASE		P VALUE
		No	%	No	%	
1.	Term	165	89.7	137	82.1	0.04*
2.	Preterm	19	10.3	30	17.9	0.04*
3.	Abortion	14	7	25	12.5	0.124
4.	PROM	11	5.5	28	15.9	0.006*
5.	APH	2	1	6	3	0.284
6.	IUGR	3	1.5	10	5	0.087
7.	Still birth/ IUD	5	2.5	5	2.5	0.999

Table 5 compares the Obstetric outcome between the controls and the cases. The number of patients who reached full term among control group was 89.7% and cases were 82.1% with a p value of 0.04 which is statistically significant . The number of patients who delivered preterm among control group was 10.3% and among cases

were 17.9% with a p value of 0.04 that is statistically significant . The percentage of patients who had PROM among control group were 5.5 % and among cases were 14% with a p value of 0.006, which is again statistically very significant

Fig 3: comparison of obstetric outcomes between the groups

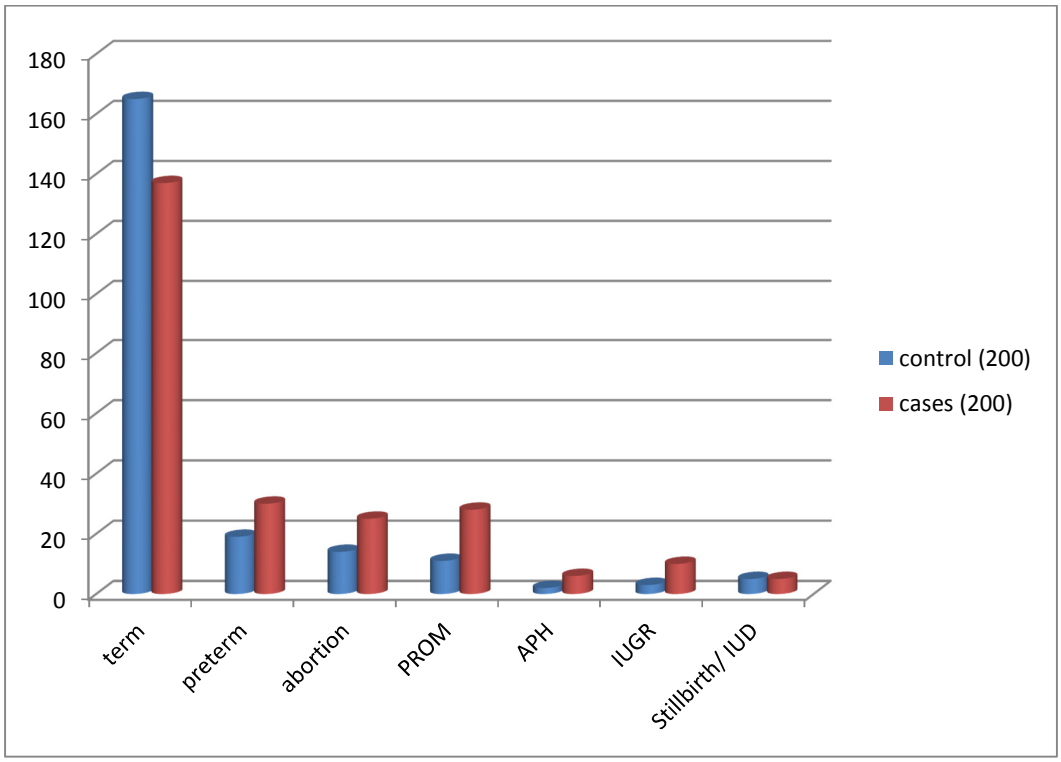


Table 6: Mode of termination of pregnancy

MODE OF TERMINATION OF PREGNANCY	CONTROL (n = 200)		CASE (n=200)		P VALUE	STATISTICAL TEST
	NO	%	NO	%		
Abortion	14	7	25	12.5	0.124	Fisher's Exact test
Normal vaginal delivery	138	69	82	41	<0.0001*	Chi square test
Assisted vaginal delivery	8	4	4	2	0.38	Fisher's Exact test
LSCS	40	20	89	44.5	<0.0001*	Chi square test

Table 6 compares the mode of termination of pregnancies between the cases and the controls. 69% of controls had normal deliveries and only 41 % of cases had normal deliveries. 20 percent of controls had LSCS and 44.5% of cases had LSCS. 4 % of controls had assisted vaginal delivery, and 2 % of cases had assisted vaginal delivery. 7 % of controls had early miscarriage and 12.5% percent of cases had early miscarriage.

Figure 4: Mode of termination of pregnancy

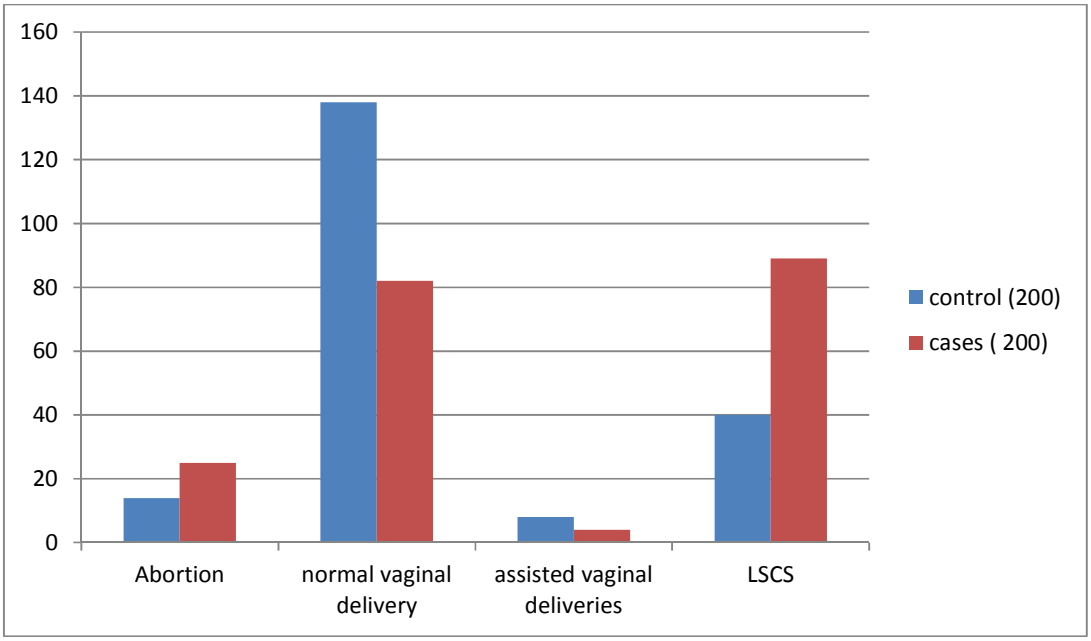


Table 7: Indication for LSCS

S. No	Indication for LSCS	Control (n=40)	Cases (n=88)
1	CPD	6 (15%)	12 (13.6%)
2	Fetal Distress	6(15%)	14 (15.9%)
3	PROM/Failed induction	4 (10%)	12 (13.6%)
4	GHT/Failed induction	3 (7.5%)	13 (14.7%)
5	Prolonged pregnancy/ failed induction	5 (12.5%)	0 (0%)
6	Severe oligohydraminos /Failed induction	4 (10%)	4 (4.5%)
7	IUGR with Fetal Distress or abnormal Doppler	2 (5%)	5 (5.68%)
8	Placenta previa	0 (0%)	4 (4.5%)
9	Abruption / unfavourable cervix	2 (5%)	5 (5.68%)
10	Severe Pre-eclampsia/ unfavourable cervix	3 (7.5%)	2 (2.27%)
11	AP Eclampsia/ unfavourable cervix	4 (10%)	6 (6.8%)
12	Others	1 (2.5%)	11 (12.5%)

Table 7 shows the number of patients who have undergone LSCS in case group is more (44.5%) compared to the control group (20%) with a p value of <0.0001. The commonest indication for LSCS in the control group is CPD (15%) and fetal distress(15%) and in the case group is fetal distress (15.9%) and GHT / failed induction (14.7%).

Fig 5: Indication for LSCS

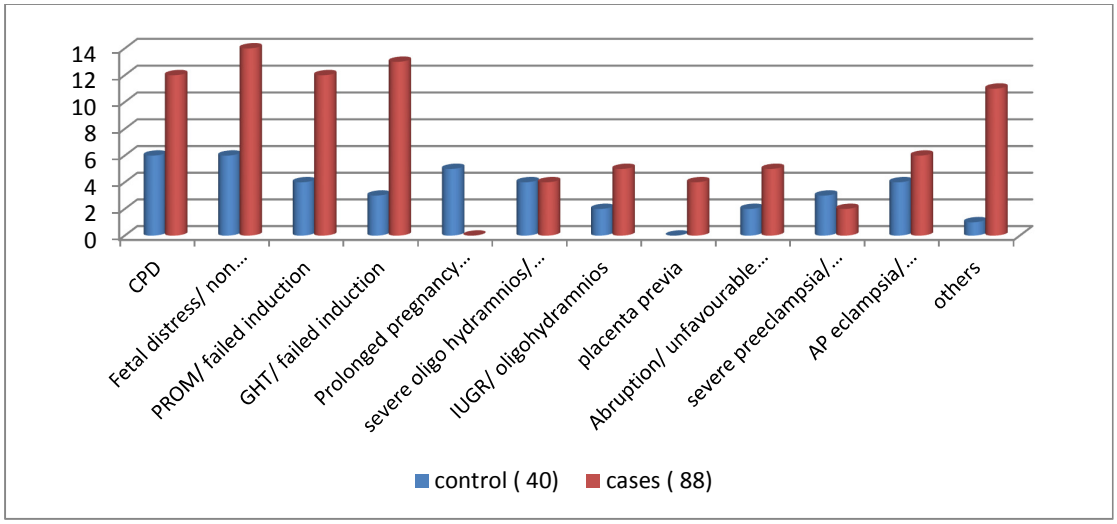


Table 8: Description of frequency of occurrence of complications between the two groups

S. No	Type of complications	Control (n=200)	Cases (n=200)
1	GHT	9 (4.5%)	20 (10%)
2	Severe pre-eclampsia	8 (4%)	5 (2.5%)
3	AP eclampsia	2 (1%)	5 (2.5%)
4	Placenta previa	6 (3%)	4 (2%)
5	Abruption	3 (1.5%)	5 (2.5%)
6	GDM	4 (2%)	10 (5%)
7	Hypothyroidism	4 (2%)	13 (6.5%)
8	Postpartum Hemorrhage	4 (2%)	2 (1%)

Table 8 describes the most common complications that are found in both control group and case group. GHT (10%), GDM (5%) and Hypothyroidism (6.5%) are found to be common among women with history of recurrent pregnancy loss.

Fig 6: Comparison of complications between the groups

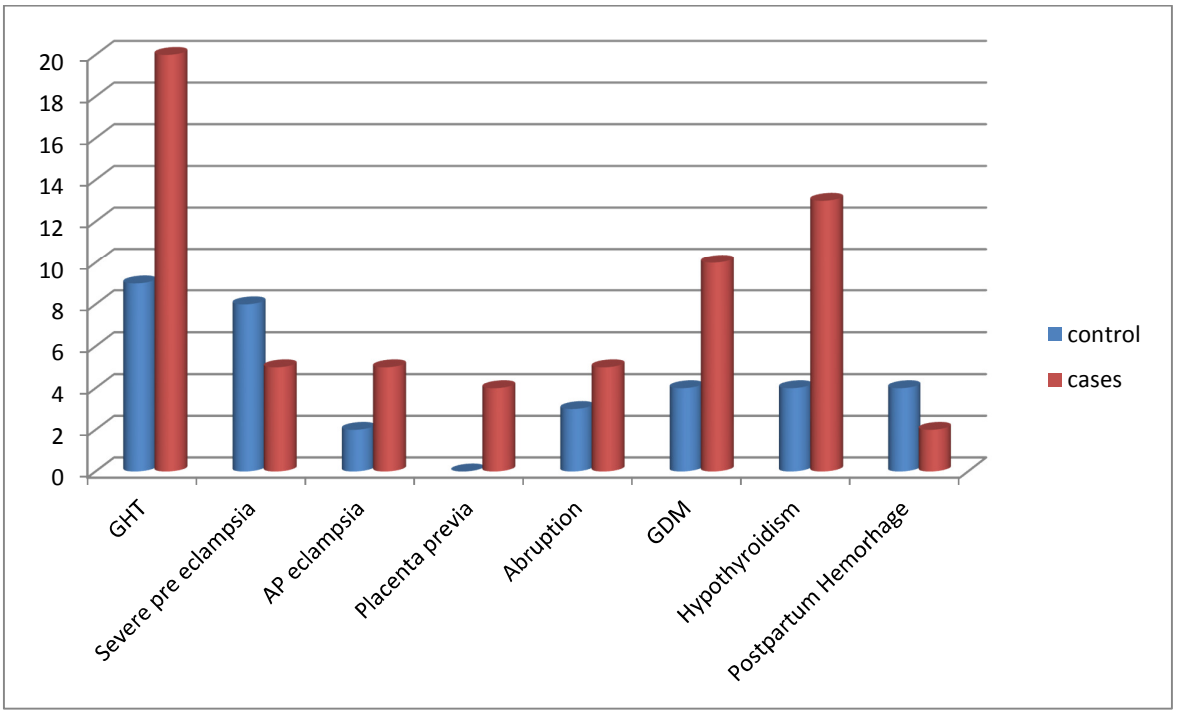


Table 9 : Comparison of neonatal outcomes between the groups

S.NO	Neonatal outcome	Control		Cases	
		No	%	No	%
1	Liveborn	180	96.7	172	98.2
2	Deadborn	6	3.3	3	1.8
	Total deliveries	186	100	175	100

Table 9 describes the neonatal outcome between the groups. Of the total 186 deliveries in the control group 180 women (96.7%) had live birth and 6 (3.3%) had dead born. Total number of deliveries in women with recurrent pregnancy loss is 175 of which 172 (98.2%) women had live birth and 3 (1.8%) had dead born.

Figure 7: Comparison of neonatal outcomes between the groups

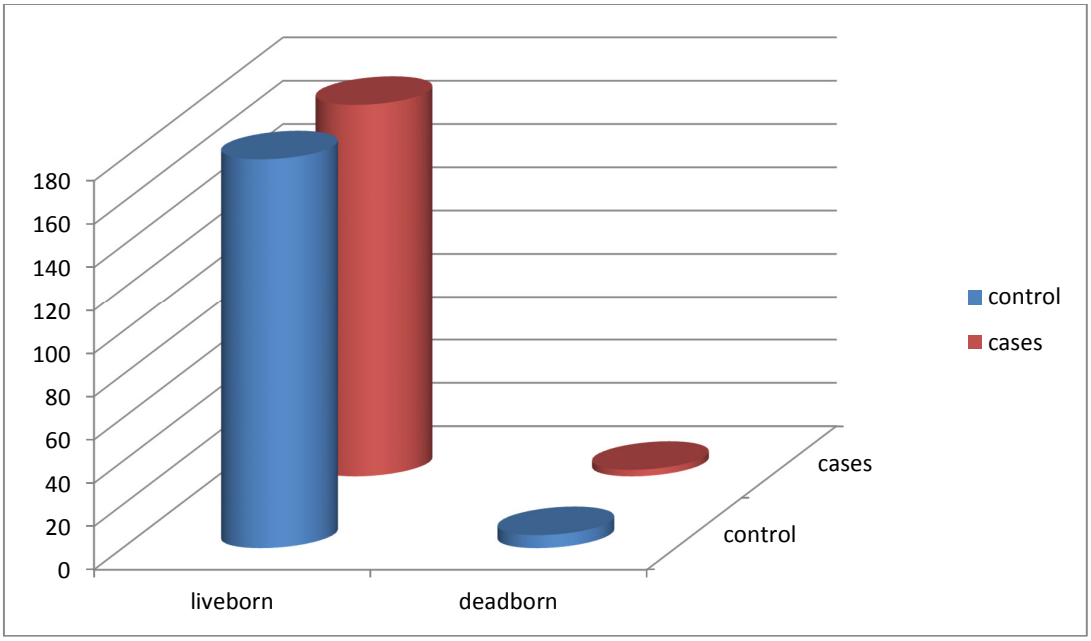


Table 10 : Comparison of fetal maturity outcomes between the groups

S. No	Parameter	Control (n=180)	Cases (n=173)	P value	Statistical test
1	Pre Term	19 (10.3%)	30 (17.9%)	0.04*	Fisher's Exact test
2	Term	166 (89.7%)	145(82.1%)	0.04*	

Of the total 180 live births in the control group, 19 babies (10.3%) were born preterm and 165 babies (89.7%) were born term .With total 172 livebirths in case group, 30 (17.9%) were born preterm and 145 (82.1%) were born term. Comparison of preterm birth between the controls and cases were statistically significant with a p value of 0.04 .

Fig 8: Comparison of fetal maturity between groups:

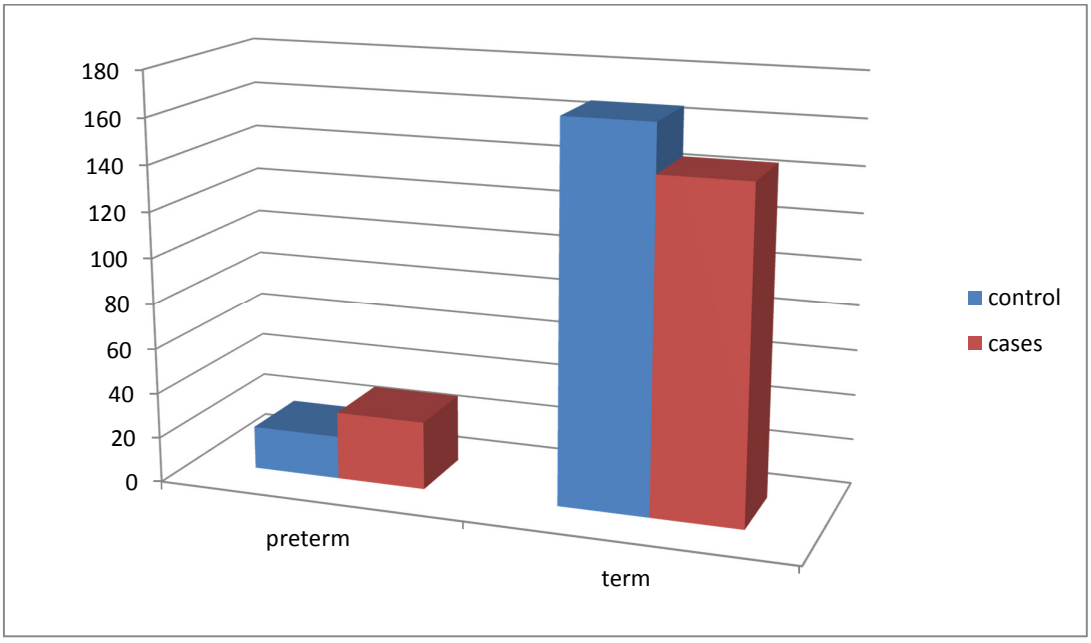


Table 11 : Comparison of APGAR score between the two groups

S. No	Apgar score	Control (n=180)	Cases (n=172)	P value	Statistical test
1	Overall score mean with SD	7.64 ± 1.37	7.2 ± 1.9	0.011*	Unpaired t test
2	Median and Mode score	8	8	-------	
Frequency distribution of the Apgar score					
S.No	Score value	Control group (n=180)	Case group (n=172)		
3	Score-4	5 (2.64%)	0 (0%)		
4	Score - 5	2 (1.05%)	12 (6.77%)		
5	Score -6	7 (3.7%)	8 (4.51%)		
6	Score -7	33 (19.5%)	47 (28.3%)		
7	Score-8	108 (59.78%)	94 (54.3%)		
8	Score -9	25 (13.22%)	11 (6.2%)		

(Data are expressed as mean ± SD for overall score and as absolute numbers with percentage for others. * indicates $p<0.05$ and considered as statistically significant.)

Table 11 describes the comparison of APGAR score between the two groups which is statistically significant with a p value of 0.011. 25 babies (13.2%) in control group and only 11 babies (6.2%) in the case group had APGAR score of 9 and more. The mean APGAR score in control group is 7.6 and that of cases is 7.2.

Table 12: Description of birthweight of the neonates between the groups

S. No	Birth weight range in Kg	Control (n=180)	Cases (n=173)
1	Less than 2.5 Kg	32 (17.3%)	64 (36.4%)
2	Between 2.5 to 3 kg	101 (58.1%)	69 (42.3%)
3	Greater than 3 Kg	47 (24.6%)	40 (21.3%)

Table 12 describes that among the primigravidae , the incidence of low birth weight (< 2.5kg) is 17.3 % whereas in women with history of recurrent pregnancy loss, the incidence of low birth weight is found to be increased (36.4%). This outcome is comparable to other studies by Eva Alberman et al , Joaquin.Paz et al , where they concluded that mean birth weight of babies preceding a spontaneous fetal loss was lower than that of pregnancy following previous live births.

Fig 9: Comparison of birthweight between the two groups

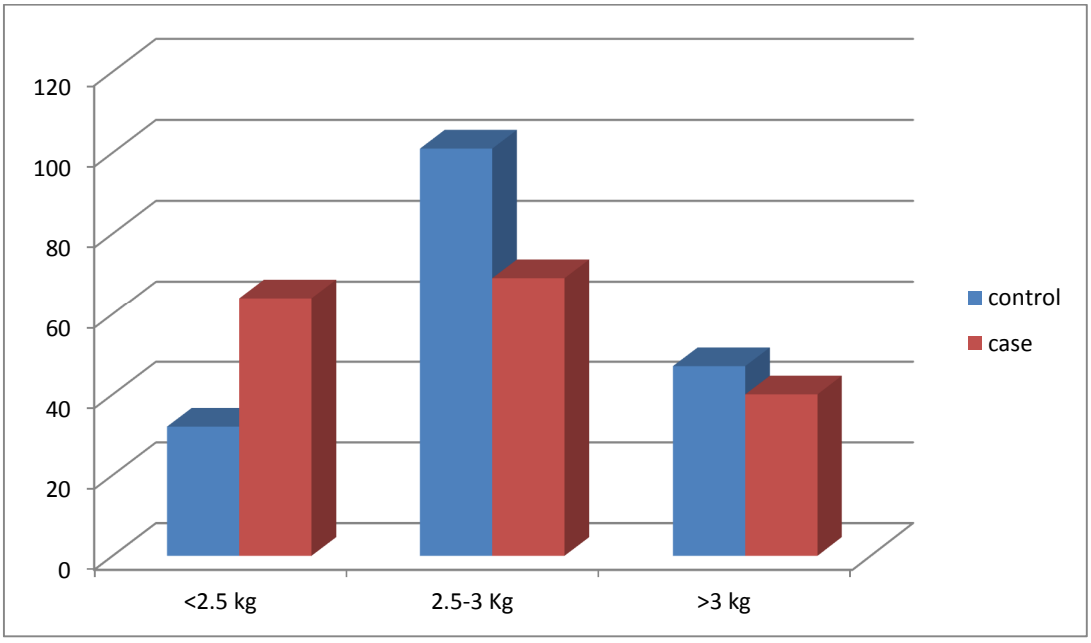
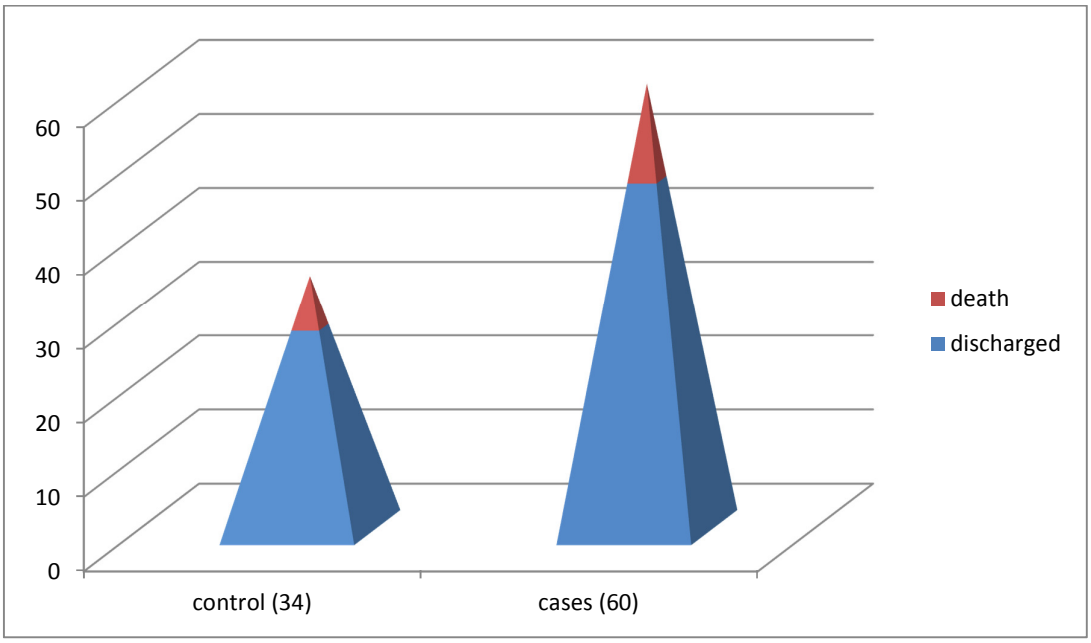


Table 13: Comparison of NICU admission and neonatal death between the groups .

S. No	Neonatal outcome	Control	Cases
	Total NICU admission	34 (18.8%)	60 (34.6%)
1	Discharged	27(79.4%)	47(78.3%)
2	Death (in NICU)	7 (20.6%)	13 (21.6%)

Table 13 compared the number of babies that got admitted in NICU in both control and cases group. Of the 180 live births in control group, 34 babies (18.8%) got admitted and 7 of these babies died in NICU. Of the 173 live births in case group, 60 babies (34.6%) got admitted in NICU and 13 of them died .

Fig 10: comparison of NICU admission between the groups



STATISTICAL METHODS

Chi-square and Fisher Exact test has been used to test the significant proportion of study characteristics between two groups.

1.Chi-Square Test

$\chi^2 = \frac{\sum (Oi - Ei)^2}{Ei}$, Where Oi is Observed frequency and Ei is Expected frequency

2. Fisher Exact Test

Fisher Exact Test statistic= $\sum p = \frac{(a+b)!(c+d)!(a+c)!(b+d)!}{n!} \frac{1}{\sum a!b!c!d!}$

3. Significant figures

Suggestive significance 0.05<P<0.10

Moderately significant 0.01<P <0.05

Strongly significant P< 0.01

Statistical software:

The Statistical software namely SPSS 11.0, Stata 8.0,Systat 11.0, Medcalc 9.0.1 and Effect Size calculator were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc.

DISCUSSION

There were 200 cases of women with history of recurrent pregnancy loss in this study. There were 200 controls comprised of primigravidae who delivered subsequent to in this study. The outcome has been analysed with respect to the following factors

1. Age of the patient
2. Occurrence of complications
3. Obstetric outcome
4. Mode of termination of pregnancy
5. Indication for LSCS
6. Neonatal outcome
7. APGAR score
8. Birth Weight
9. NICU admission

Table 3: Out of the 200 cases and 200 controls. 60 % belonged to 21-25 years of age among the control group and among controls only 22% of patients belonged to the same age group. 31% belonged to 26-30 years

among control group and 69 % belonged to 26 to 30 years among cases group .The age distribution among the cases differ from that of the controls. Women with history of recurrent pregnancy loss has comparatively higher age group than the controls. This may be due to the fact that women with higher maternal age have increased risk of pregnancy loss.

According to M.J.N.C Keirse et al, advanced maternal age is being strongly associated with several adverse maternal and perinatal outcomes.[89] Another reason for this higher age group among the cases is almost 30 % of women with history of recurrent pregnancy loss in this study had history of taken treatment for infertility.

According to study by Brigham et al (1999) increasing maternal age reduces the chance of a pregnancy success.[86] Although the confidence intervals for the success prediction are wide at the extreme ends of the age spectrum, there is little doubt that maternal age has a significant impact on future success in the recurrent miscarriage population.

Table 6: Distribution of case group with respect to the number of previous abortions.

Out of the 200 cases studied, 84.5% of patients had history of 2 previous abortions, 13 percent of patients had previous 3 abortions, 1.5 percent of patients had 4 previous abortions and 1 percent of patient had previous 5 or more abortions.

S.No	No. of abortions	Our study (%)	Brigham et al[86] (1999) (%)
1	2	84.5	24
2	3	13	48
3	4	1.5	13
4	5	1	14

Table 7 compares the Obstetric outcome between the controls and the cases. The number of patients who reached full term among control group was 89.7% and cases were 82.1%. Ekwo EE et al (1993) in their study of 176 women enrolled showed that percentage of women having prior abortion had less number of patients reaching to successful outcome.[92]

S.No	Study	% of women who reached full term
1.	Our study	68.5%
2.	Costa et al (2015)[93]	75%
3.	Tanya Pradhan et al (2015)[94]	71.9%
4.	Agrawal S et al (2015)[82]	74.3%

The number of patients who delivered preterm among control group was 10.3% and among cases were 17.9% which was statistically significant with p value of 0.04. Similar study done by P.W.Reginald, R.W.Beard (1987) showed that 28 percent of patients having preterm with >3 abortion in the previous pregnancies.[87] Olga Basso et al showed that there is increased incidence of preterm births in abortion cohort as compared to the reference cohort.[95]

Similarly studies by David H Thom et al, MJNC keirse et al, RW Rush et al, Agrawal et al (2015) showed increased incidence of preterm birth in women with history of recurrent pregnancy loss. In a study by David H Thom et al (1992), adjusted odd ratio for preterm birth is 2.6 (95% CI, 1.7-4) and Makhlaouf MA et al (2014) showed a relative risk of 1.5 (95% confidence interval 1.1-2.1).[83,96]

S.NO	Study	Incidence of preterm birth
1	Our study	17.9 %
2	Reginald PW et al (1987)[87]	28%
3	Jivraj et al (2001)[97]	13%
4.	Tanya Pradhan et al(2015)[94]	18.5%
5.	Gabbai D et al (2017)[98]	22.9%
6.	Agrawal et al (2015)[82]	-

The percentage of patients who had PROM among control group were 5.5 % and among cases were 14% which is statistically significant with a p value of 0.006. A study by David H Thom et al evaluated the association of history of spontaneous abortion and subsequent adverse birth outcomes. Similar to our study, this study has also showed increased incidence of

PROM in women with recurrent pregnancy loss. Similar study done by Edem E.Ekno et al showed in his study "Previous pregnancy outcome and subsequent risk to preterm rupture of amniotic sac membranes" that previous preterm delivery, abortion and prematurity all increase the risk for subsequent preterm birth with / without PROM.[92] The Risk of PROM in women with ecurrent pregnancy loss were evaluated in the studies by Makhlouf MA et al (2014) (OR 2.9; 95% CI, 1.6-5.3) and David H Thom et al (1992) (RR – 1.8, 95% CI, 1.2 – 2.9).[83,96]

Percentage of patients having IUGR in our cases group were 5% and which was more than 1.5% among the control group, but there was no statistical difference between these two (P=0.087). The studies done by David.K.Thomas et al showed that risk of IUGR and low birth weight increases as the number of abortions increases.[83] With one prior spontaneous abortions 4.9 % and with three or more prior spontaneous abortions it was 9.5%.

S.No	Study	Incidence of IUGR
1.	Our study	5%
2	Tanya Pradhan et al (2015)[94]	12.8%
3.	Jing Yang (2017)[98]	11%
4.	Agrawal et al (2015)[82]	-

Table 6 compares the mode of termination of pregnancies between the cases and the controls. 69% of controls had normal deliveries and only 41 % of cases had normal deliveries which is statistically significant with a p value of 0.0001.

20 % of controls had LSCS and 44.5 % of cases had LSCS which is also statistically significant with a p value of 0.0001. In a study by Agrawal S et al (2015), they concluded that there is increased need for caesarean section (23.3%) in cases of previous caesarean sections.[82] 4 % of controls had assisted vaginal delivery, and 2 % of cases had assisted vaginal delivery.

S.No	Study	Incidence of LSCS	P Value
1	Our study	44%	<0.0001
2.	Jivraj et al(2001)[96]	36%	<0.05
3.	Sheiner E et al (2005)[91]	15.9%	<0.001
4.	Agrawal S et al (2015)[82]	23.3%	-
5.	Jing yang et al (2017)[98]	22%	<0.001
6	Tanya Pradhan et al(2015)[94]	-	-

7 % of controls had early miscarriage and 12.5% of cases had early miscarriage. This outcome is comparable to the study by Agrawal S et al (2015) , in which the incidence of abortion was found to be 14.3 % .[82] This is further supported by S A Brigham et al who showed a decrease in pregnancy success rate with increasing number of previous abortion. In a study by Ulla Breth Knudsen et al , the overall risk of spontaneous abortion was 11% and the risk was 16, 25, 45 and 54% after 1 to 4 previous consecutive spontaneous abortions .[90]

S.No	Study	Incidence of abortion
1.	Our study	12.5%
2.	Agrawal S et al (2015)[82]	14.3%
3.	Ulla breth Knudsen et al [90]	11%
4.	Jing Yang et al (2017)[98]	16%
5.	Tanya Padhan et al(2015)[82]	-

Table 7 shows that the commonest indication for LSCS in the control group is Cephalopelvic disproportion (15%) and fetal distress(15%) and in the case group is fetal distress (15.9%) and GHT / failed induction (14.7%). Though the incidence of LSCS is more in women with history of recurrent pregnancy loss, the indications for LSCS and their proportion is almost similar in both groups .

Table 8 describes the most common complications that are found in both control group and case group. In our study, we found that GHT (10%), GDM (5%) and Hypothyroidism (6.5%) are more common among women with history of recurrent pregnancy loss. Other pregnancy complications like IUGR(5%), placenta previa (2%), Abruption (2.5%) were also found to be increased in case group though not significant. Similarly a study by Eyal Sheiner & Amalia Levy et al, pregnancy complications such as hypertensive disorders , abruption, IUGR and caesarean section were found to be in a higher rate in women with recurrent abortions.

S.NO	Study	Incidence of complications			
		GHT	Preeclampsia	Placenta Previa	Abruption
1.	Our study	4.5%	4%	3%	1.5%
2.	Tanya Pradhan et al (2015)[94]	5.7%	5.7%	-	4.2%
3.	Jing Yang et al (2017)[98]	17%	24%	21%	3%
4.	David H Thom et al (1992)[83]	-	-	6%	-

Table 9 & 10 describes the neonatal outcome between the groups.. Total number of deliveries in women with recurrent pregnancy loss is 175 of which 172 (98.2%) women had live birth and 3 (1.8%) had dead born. Overall the incidence of term live birth in our study is 68.5%. This is similar to the study by Agrawal et al(2015) , the incidence of term live birth was found to be 74.3%.

Table 11 describes the comparison of APGAR score between the two groups which is statistically significant with a p value of 0.011. The mean APGAR score in control group is 7.6 and that of women with recurrent pregnancy loss is 7.2. This was similar to the study by Mona Fawzy et al (2016) showed that women with recurrent miscarriage had significantly increased odds of low Apgar scores at one (odds ratios (OR) 1.57, 95% CI 1.20–2.05) and five minutes (OR 2.0, 95% CI 1.23–3.27).

Table 13 compared the number of babies that got admitted in NICU in both control and cases group . 18.8 % of the babies of control group were admitted in NICU and 34.6 % of the babies of case group got admitted which is very high. The most common cause of admission for these babies are low birth weight and prematurity.

Nearly 50% of Preterm Neonates required NICU admission due to various causes whereas other 50% needed only routine neonatal care. Amongst Term Neonates only 14% required NICU admission and 78.3% were shifted to mother side and given routine neonatal care.

SUMMARY

1.Previous history of recurrent pregnancy loss increases the risk of adverse maternal and perinatal outcome in the future pregnancies.

2. There is association between previous recurrent abortion and preterm delivery(17.9%), Recurrence of abortion(12%) and PROM (14%), in the subsequent pregnancies.

3.There is no statistically significant increase in the rate of IUGR, APH, still birth, IUD in the subsequent pregnancies.

4. There is increased need of caeserean sections for various indications in women with recurrent pregnancy loss. The incidence of LSCS in these women in our study is 44%.

5. History of recurrent pregnancy loss has been associated with low birth weight of the babies in subsequent pregnancy. 36.4 % of the babies of these women in our study have low birth weight (<2.5 kg).

CONCLUSION

RPL is an important reproductive health issue. Various etiologies have been identified over the years and successful therapeutic strategies implemented. A full workup can be initiated following two consecutive losses to identify treatable causes that include uterine abnormalities, APS, endocrine diseases, and balanced translocations. Lifestyle modifications should also be implemented to improve reproductive prognosis. However, almost half of the cases remain unexplained, for which various treatments are continuously being developed. Regardless of the cause, a thorough follow-up with an important psychological support can help most couples achieve a successful live birth. Hence careful surveillance is required in pregnancies following recurrent abortions, for early detection of possible complications.

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PROFORMA

Name: Age: IP.No:

Occupation : Address:

Date of admission: Date of Discharge:

MENSTRUAL HISTORY:

Age at Menarche:

Cycles: Regular/Irregular:

LMP:

MARITAL HISTORY:

Married for

Consanguinity:

Treatment for infertility

OBSTETRRIC CODE:

PREVIOUS OBSTETRIC HISTORY:

PRESENT PREGNANCY:

LMP: **EDD:**

1st trimester

2nd trimester

3rd trimester

PAST HISTORY: Diabetes / hypertension/ renal disease/ SLE/ rheumatoid arthritis/ cardiac disease/ asthma / epilepsy/ others

SURGICAL HISTORY:

FAMILY HISTORY:

PERSONAL HISTORY:

HUSBAND:

Age:

Occupation:

Smoking/alcohol:

GENERAL EXAMINATION:

Height:

Weight : BMI :

Breast:

Thyroid :

BP : Pulse: Cvs/Rs:

P/A:

P/V:

INVESTIGATIONS

Hb%

Urine Routine

Blood sugar:

S.urea:

S.creatinine:

Blood group:

VDRL:

HIV:

HBsAg:

Pelvic Ultrasonogram:

SPECIFIC INVESTIGATIONS:

Glucose Tolerance Test:

Thyroid Stimulating Hormone:

APLA screening (if done)

Obstetric outcome:

Abortion:	Spontaneous expulsion:
Preterm:	PROM
Term:	Post term:
IUGR:	IUD/STILL BIRTH:

MODE OF DELIVERY:

Normal vaginal delivery:	Assisted breech delivery:

Assisted deliveries:

Duration of labour:

LSCS:	Indication:

BABY DETAILS:

Sex: Weight: Apgar score:

NICU Admission:

Outcome:

COMMENTS:

PATIENT CONSENT FORM

Study Detail: A Study on "MATERNAL AND PERINATAL OUTCOME IN PREGNANCY FOLLOWING RECURRENT PREGNANCY LOSS"

Study Centre: Department of Obstetrics & Gynaecology, Thanjavur medical college, Thanjavur.

I confirm that I have read and understood the information Sheet for the above study. I have had the opportunity to ask questions and all my questions and doubt have been answered to my complete satisfaction.

I understand that my participation in the study is voluntary and that i am free to withdraw at an time, without giving any reason, without my legal rights being affected.

I understand that the Clinical study personnel, the Ethics Committee and the Regulatory Authorities will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if i withdraw from the study. I agree to this access. However, I Understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

I agree to take part in the above study and to comply with the instructions given during the study and to faithfully co-operate with the study team, and to immediately inform the study if I suffer from any deterioration in my health of well being or any unexpected or unusual symptoms.

I hereby give permission to undergo completed clinical examination and diagnostic tests including haematological, biochemical, radiological tests.

I hereby consent to participate in this study.

Signature/Thumb impression:..... Place Date of the patient

Patient's Name, Address &Ph.No:.....

Name of the Investigation:.....

Signature of the Investigator: ..... Place.....Date

Institution : .....

Signature of the Relative/Gardian.....

MASTER CHART

.	Name	Age	IPNo	Booking Status	Menstrual Histroy	Marital H/o (YEARS)	CONSANGUINITY	Obstetric H/o	Gestational Age (WEEKS)	OBSTETRIC OUTCOME						MODE OF DELIVERY			INDICATION FOR ISCS														COMPLICATIONS								
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	ANTEPARTUM								
																																GHIT	Severe Preeclampsia	AP eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm
1	Sudha	22	465918	B	R	1	NCM	Primi	37							✓																								
2	Geetha	23	466002	B	R	1	NCM	Primi	40			✓						✓												✓									
3	Sivaranjani	21	466635	UB	R	2	NCM	Primi	39							✓																						
4	Kalairani	24	465693	B	IR	1	CM	Primi	39							✓																						
5	Reethika	22	466550	UB	R	1	CM	Primi	29		✓					✓																	✓					✓
6	Amudha	30	465078	UB	R	1	NCM	Primi	39									✓						✓													
7	Kasthuri	21	467209	B	R	2	CM	Primi	37							✓																						
8	Kiruthiga	26	467572	B	IR	3	NCM	Primi	39							✓																						
9	Kamatchi	30	467609	B	R	1	NCM	Primi	37							✓																					✓	
10	Malathi	26	468261	UB	R	1	NCM	Primi	40							✓																						
11	Saranya	24	467689	B	IR	1	NCM	Primi	40									✓	✓																		
12	Karthika	25	468479	B	R	1	NCM	Primi	39							✓																						
13	Ayshambal	21	468769	B	R	1	NCM	Primi	40							✓																						
14	Jeya Bharathi	29	469122	UB	R	1	NCM	Primi	39									✓										✓					✓					
15	Nalini	23	469175	B	R	1	NCM	Primi	37						✓			✓											✓				✓					
16	Durgadevi	23	469282	B	IR	1	NCM	Primi	37								✓																				
17	Amsavalli	29	469732	UB	IR	7	NCM	Primi	40									✓						✓													
18	Nadhiya	22	469780	B	R	3	NCM	Primi	37							✓																						
19	Sathiya	27	469730	B	IR	1	CM	Primi	37							✓																						
20	Syeda	29	469846	UB	R	2	NCM	Primi	37									✓										✓					✓					
21	Gayathri	21	470192	B	R	1	NCM	Primi	40							✓																						
22	Poongodi	28	442172	B	R	1	NCM	Primi	39							✓																						
23	Thenmozhi	27	442455	B	R	1	NCM	Primi	41									✓	✓																		
24	Nadhiya	30	442617	B	R	1	NCM	Primi	33		✓					✓																					✓
25	Jeevitha	25	443418	UB	R	2	NCM	Primi	40							✓																						
26	Suguna	24	443415	B	R	1	1 CM	Primi	41									✓																				
27	Revathy	32	443017	B	IR	1	NCM	Primi	8	✓																												
28	Sathiyabama	22	443145	B	R	1	NCM	Primi	40									✓					✓															
29	Prabhavathy	24	441640	B	R	1	NCM	Primi	39							✓																						
30	Mirmala	24	443178	B	R	2	NCM	Primi	38							✓																						
31	Jeyamani	27	442413	B	R	1	CM	Primi	41																								✓							
32	Ramathilagam	25	443148	UB	R	1	NCM	Primi	39						✓	✓																				✓			
33	Ganga	26	442047	B	IR	3	NCM	Primi	39							✓																						

.	Name	Age	IPNo	Booking Status	Menstrual Histroy	Marital H/o (YEARS)	CONSANGUINITY	Obstetric H/o	Gestational Age (WEEKS)	OBSTETRIC OUTCOME						MODE OF DELIVERY																								COMPLICATIONS								
																			INDICATION FOR ISCS																			ANTEPARTUM									
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHIT	Severe Preeclampsia	AP eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm
34	Ambigai	30	444184	UB	R	1	NCM	Primi	38							✓																														
35	Sathya	35	443372	B	R	1	NCM	Primi	37									✓	✓																✓												
36	Maheswari	22	443461	B	IR	1	CM	Primi	39									✓	✓																												
37	Suganya	29	454502	B	R	2	CM	Primi	40							✓																														
38	Madhamitha	27	456143	B	R	1	NCM	Primi	39							✓																														
39	Oorvasi	25	454530	B	R	1	NCM	Primi	41							✓																														
40	Dhanalaksmi	25	452816	UB	R	3	NCM	Primi	38																																				
41	Jenifer Esther	26	452331	B	IR	1	NCM	Primi	27	✓																																				✓
42	Kavitha	24	453149	B	R	2	NCM	Primi	40									✓		✓																										
43	Kamala	22	453470	B	R	1	NCM	Primi	38							✓																														
44	Rabiya Begum	24	453784	B	R	2	NCM	Primi	38					✓				✓									✓																				
45	Annakodi	28	457401	B	R	1	NCM	Primi	38																																				
46	Leelavathy	26	452617	B	R	1	NCM	Primi	35		✓					✓																														✓
47	Chandra	27	456349	B	IR	5	NCM	Primi	36		✓					✓																														✓
48	Kameshwari	24	456234	B	R	1	NCM	Primi	39																																				
49	Rasiya	25	458291	UB	R	1	NCM	Primi	39							✓																														
50	Punitha	23	458014	B	R	1	CM	Primi	40			✓				✓																														
51	Kamachi	26	458143	UB	IR	1	NCM	Primi	9	✓																																				
52	Nasriya Thasneem	26	458343	B	R	1	NCM	Primi	37				✓		✓			✓									✓																				
53	Boomika	31	457177	B	R	1	CM	Primi	38							✓																														
54	Eshwari	27	457188	B	R	1	NCM	Primi	39							✓																														
55	RamyaKumari	37	457001	B	R	1	NCM	Primi	40							✓																														
56	Kavitha	27	457807	B	IR	1	NCM	Primi	39			✓						✓			✓																										
57	Parameshwari	27	457719	B	R	1	NCM	Primi	41									✓					✓																								
58	Saradhammal	23	457560	UB	R	7	CM	Primi	40							✓																														
59	Anthony mary	25	458521	B	R	1	NCM	Primi	38							✓																														
60	Yogeshwari	26	458018	B	R	1	NCM	Primi	39							✓																														
61	Kodeeshwari	23	458124	B	R	1	NCM	Primi	41							✓																														
62	Palaniyammal	27	451991	B	R	1	NCM	Primi	10	✓																																				
63	Punith Lakshmi	26	452047	B	R	1	NCM	Primi	37							✓																														
64	Mahamrth nisha	26	452157	B	R	1	NCM	Primi	38							✓																														
65	Savariyaval	25	452477	B	IR	4	CM	Primi	38							✓																														
66	Manonmani	27	452687	B	R	1	NCM	Primi	40							✓																														
67	Thara	28	452806	B	IR	1	NCM	Primi	38			✓				✓																														
68	Selvarani	24	452917	UB	R	1	NCM	Primi	39							✓																														
69	Indhurekha	24	453105	B	R	1½	NCM	Primi	40									✓	✓																											
70	Amirtha lakshmi	25	453269	B	R	2	NCM	Primi	37							✓																														
71	Umapathy	27	453340	UB	IR	2	CM	Primi	38							✓																														
72	Nandhini	27	453518	B	R	1	NCM	Primi	7	✓																																				

.	Name	Age	IPNo	Booking Status	Menstrual Histroy	Marital H/o (YEARS)	CONSANGUINITY	Obstetric H/o	Gestational Age (WEEKS)	OBSTETRIC OUTCOME						MODE OF DELIVERY			INDICATION FOR ISCS														COMPLICATIONS								
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	ANTEPARTUM								
																																GHIT	Severe Preeclampsia	AP eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm
73	Vetriselvi	25	453720	B	R	1	NCM	Primi	35		✓					✓																							✓
74	Karthiga	29	453888	B	R	1	NCM	Primi	37							✓																	✓							
75	Kamaleshwari	25	453987	B	R	3	NCM	Primi	39								✓																						
76	Loogeshwari	24	454807	B	R	1	NCM	Primi	39							✓																				✓	✓			
77	Bhuvaneshwari	25	454317	B	IR	1	CM	Primi	11	✓																													
78	Rabiyathul Bashiya	27	454530	B	R	1	CM	Primi	41							✓																							
79	Shanthi	26	454618	B	R	1	NCM	Primi	40							✓																							
80	Sangeetha	25	454821	B	R	1	NCM	Primi	40									✓		✓																			
81	Vinitha	25	454923	B	R	1	NCM	Primi	40							✓																							
82	Sylvia	27	454102	UB	R	8	NCM	Primi	37					✓				✓											✓							✓				
83	Deepika	24	454204	UB	IR	1	NCM	Primi	38							✓																							
84	Manohari	25	455189	B	R	1	NCM	Primi	28		✓																												✓
85	Padma	26	455273	B	R	1	CM	Primi	38							✓																							
86	Veena	32	455380	B	IR	1	NCM	Primi	36		✓					✓																							✓
87	Mohana	30	455417	UB	R	4	NCM	Primi	40							✓																							
88	Gayathri	25	455521	B	R	1	NCM	Primi	40							✓																							
89	Ramathilagam	25	455690	B	R	1	NCM	Primi	38							✓																							
90	Vartheeshwari	25	455802	UB	R	1	NCM	Primi	38									✓								✓						✓							
91	Ragasiya	27	455936	B	R	1	NCM	Primi	10	✓																												
92	Kumudha	27	456117	B	IR	1	NCM	Primi	39							✓																							
93	Vasantha	25	456298	B	IR	1	CM	Primi	38							✓																							
94	Priyadarshini	23	456410	UB	R	1	NCM	Primi	40							✓																							
95	Subiksha	33	456489	B	R	3	NCM	Primi	41									✓					✓															
96	Narmatha	25	456547	B	R	1	CM	Primi	38							✓																							
97	Suganya	27	456580	UB	R	1	NCM	Primi	39							✓																							
98	Jeevitha	29	456714	B	R	1	NCM	Primi	40									✓			✓																	
99	Veerakumari	28	456811	B	IR	2	NCM	Primi	40							✓																							
100	Samsath Begam	28	456892	B	R	1	CM	Primi	28																														✓
101	Rajeshwari	27	456912	B	R	1	NCM	Primi	24		✓					✓																							✓
102	Kaviyarasi	34	457106	B	R	1	NCM	Primi	37							✓																							
103	Thilagavathi	33	457280	B	IR	2	NCM	Primi	36		✓					✓																							✓
104	Umadevi	24	467416	B	R	2	NCM	Primi	37									✓				✓										✓							
105	Meenakumari	27	467612	B	R	1	NCM	Primi	38							✓																							
106	Vadivukarasi	27	467704	B	R	1	NCM	Primi	38							✓																							
107	Megala	28	467816	B	R	1	NCM	Primi	26	✓					✓																							✓
108	Kanaga	27	467911	B	IR	2	CM	Primi	40							✓																							
109	Amaladevi	30	468100	B	R	1	NCM	Primi	41							✓																							
110	Victoria Joseph	26	468104	UB	R	1	NCM	Primi	41							✓																							
111	Deepa	25	468167	B	R	3	NCM	Primi	39							✓																							

.	Name	Age	IPNo	Booking Status	Menstrual Histroy	Marital H/o (YEARS)	CONSANGUINITY	Obstetric H/o	Gestational Age (WEEKS)	OBSTETRIC OUTCOME						MODE OF DELIVERY			INDICATION FOR ISCS														COMPLICATIONS								
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHIT	Severe Preeclampsia	AP eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm
112	Sundhandhiradevi	20	468240	B	R	1	NCM	Primi	41							✓																							
113	Ramya	28	468412	B	R	1	NCM	Primi	40																															
114	Renugadevi	20	461308	B	R	1	NCM	Primi	41							✓																							
115	Kanaga	20	461422	UB	R	1	NCM	Primi	40							✓																							
116	Akshaya	25	461681	B	R	3	NCM	Primi	39							✓																							
117	Manimegala	28	461804	B	IR	1	NCM	Primi	35		✓							✓											✓			✓						✓
118	kamatchi	30	462277	B	R	1	NCM	Primi	38							✓																						
119	Gandhipathi	27	462579	UB	R	1	CM	Primi	40							✓																				✓			
120	Thenmozhi	28	463741	B	R	1	NCM	Primi	41								✓																					
121	Nadhiya	26	462894	B	R	1	NCM	Primi	40							✓																						
122	Vennila	25	463604	B	R	2	NCM	Primi	8	✓																												
123	Sripriya	29	464972	B	IR	1	NCM	Primi	38							✓																						
124	Kamala	27	465872	UB	R	2	NCM	Primi	30		✓					✓																						✓
125	Sharmila Banu	30	469183	B	R	1	NCM	Primi	40							✓																						
126	Babitha	32	466528	B	IR	1	NCM	Primi	41							✓																						
127	Yuvasri	28	460104	B	R	1	NCM	Primi	39			✓						✓			✓																	
128	Geetha	27	463311	B	R	4	NCM	Primi	40							✓																						
129	Madhumitha	27	465445	B	R	1	NCM	Primi	41							✓																						
130	Ratha priya	20	462550	B	R	1	CM	Primi	41							✓																						
131	Durgadevi	25	460496	B	R	2	NCM	Primi	40							✓																						
132	Srilakshmi	29	464493	B	IR	1	NCM	Primi	38			✓						✓						✓													
133	Christy	27	463269	UB	IR	1	NCM	Primi	36									✓											✓				✓					✓
134	Kamaladevi	24	460302	B	R	1	NCM	Primi	37							✓																						
135	Deivanai	26	461149	B	R	2	NCM	Primi	38							✓																						
136	Saraswathi	26	462406	B	R	1	NCM	Primi	38							✓																						
137	Thiripurasundari	28	468001	UB	R	1	NCM	Primi	39							✓																						
138	Sophia Thomas	25	461057	B	R	1	NCM	Primi	41								✓																		✓		✓	
139	Kumaraselvi	27	465526	UB	IR	1	NCM	Primi	24	✓																												
140	Manjula	24	463335	B	R	1	NCM	Primi	41									✓					✓														
141	Benita Rose	26	460741	B	R	1	CM	Primi	40							✓																						
142	Tamilselvi	27	467499	UB	R	1	NCM	Primi	38			✓				✓																						
143	Rukumani	29	461761	B	R	2	NCM	Primi	37							✓																						
144	Saranya	30	466336	B	R	1	NCM	Primi	38			✓						✓			✓																✓	
145	Ambikavathy	25	464472	B	IR	1	NCM	Primi	39									✓	✓																	✓		
146	Radhi keuvari	26	462175	B	R	1	NCM	Primi	39							✓																						
147	Anupriya	22	465725	B	R	1	NCM	Primi	41							✓																						
148	Acilandeswari	25	462228	UB	R	1	NCM	Primi	41							✓																						
149	Karpaga Sundari	27	463796	B	R	2	NCM	Primi	20	✓																												

.	Name	Age	IPNo	Booking Status	Menstrual Histroy	Marital H/o (YEARS)	CONSANGUINITY	Obstetric H/o	Gestational Age (WEEKS)	OBSTETRIC OUTCOME						MODE OF DELIVERY			INDICATION FOR ISCS												COMPLICATIONS								
																																ANTEPARTUM								
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHIT	Severe Preeclampsia	AP eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm
150	Jeavenba	30	467747	B	R	1	CM	Primi	40							✓																						
151	Sylvia Rani	28	460515	B	IR	1	NCM	Primi	41							✓																						
152	Vanitha	22	464255	B	IR	2	NCM	Primi	34		✓					✓																						✓
153	Rasiga Priya	25	461823	B	R	1	NCM	Primi	40							✓																						
154	Velmani	28	465587	UB	R	1	NCM	Primi	38				✓		✓			✓										✓				✓						
155	Harishri	30	460733	B	R	1	NCM	Primi	39							✓																						
156	Anuradha	24	468853	B	R	1	NCM	Primi	39							✓																						
157	Narmatha Devi	29	464915	UB	IR	2	NCM	Primi	38							✓																						
158	Oorvasi	29	466919	B	IR	3	CM	Primi	39							✓																						
159	Gomathy	27	463844	B	R	1	NCM	Primi	40							✓																						
160	ManoRanjani	27	465441	B	R	1	NCM	Primi	41							✓																						
161	Swepna lakshmi	29	468851	B	R	1	CM	Primi	30		✓					✓																						✓
162	Ilampirai	27	462621	UB	R	1	CM	Primi	41									✓					✓														
163	Barkavi	26	461398	B	R	1	NCM	Primi	40								✓																					

.	Name	Age	IPNo	Booking Status	Menstrual Histroy	Marital H/o (YEARS)	CONSANGUINITY	Obstetric H/o	Gestational Age (WEEKS)	OBSTETRIC OUTCOME						MODE OF DELIVERY			INDICATION FOR ISCS														COMPLICATIONS									
																																		ANTEPARTUM									
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHIT	Severe Preeclampsia	AP eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm
164	Nithya Priya	25	464320	B	R	1	NCM	Primi	41							✓																								
165	Amirtham	20	466157	B	R	3	NCM	Primi	38							✓																						✓			
166	yazhini	33	462434	B	R	1	NCM	Primi	39									✓		✓																				
167	Vetriselvi	24	462116	B	IR	1	CM	Primi	39			✓				✓																							
168	Indupriya	26	461021	B	R	1	NCM	Primi	40							✓																								
169	karhiga	28	461363	B	R	1	NCM	Primi	41							✓																								
170	Sherly jasmine	27	469777	B	R	3	NCM	Primi	40							✓																								
171	Ragavi	25	461760	B	R	2	CM	Primi	38							✓																								
172	Atirekha	29	461733	B	R	1	NCM	Primi	35		✓					✓																							✓
173	Rajeshwari	24	463675	B	R	1	NCM	Primi	38							✓																								
174	Aarthi	20	461540	B	R	2	NCM	Primi	40			✓				✓																								
175	Bavithra	28	466697	UB	R	2	NCM	Primi	37									✓				✓															✓		
176	Dhivya vadivel	28	460969	B	R	3	NCM	Primi	38							✓																								
177	Durga	27	462608	B	IR	1	NCM	Primi	38								✓																							
178	Vigneshwari	28	464230	B	R	1	NCM	Primi	37							✓																								
179	Janani	26	461722	B	R	1	NCM	Primi	39									✓																						
180	Karkuzhazhi	25	465322	B	IR	1	NCM	Primi	40									✓						✓															
181	Lavanya	25	463888	B	R	2	NCM	Primi	22	✓																												
182	Mary Shanthini	23	464088	B	R	2	NCM	Primi	41							✓																								
183	Kanimozhi	28	462213	B	R	1	NCM	Primi	12	✓																												
184	Nirmala	27	469160	B	R	1	NCM	Primi	37							✓																								
185	Priyadarshini	30	464147	B	R	1	NCM	Primi	38							✓																								
186	Radhika	25	433003	UB	R	1	NCM	Primi	38									✓				✓																	
187	Shadika Jasmine	24	470838	B	IR	3	NCM	Primi	40							✓																								
188	Tharani	27	479160	B	R	1	NCM	Primi	39								✓																							
189	Vidhya	27	477696	B	R	1	NCM	Primi	39							✓																								
190	Ushadevi	29	471697	B	R	1	NCM	Primi	38							✓																								
191	Ranjani siva	25	472211	B	R	3	NCM	Primi	16	✓																												
192	Pre	30	477032	B	R	1	NCM	Primi	41							✓		✓																					
193	Neelaventhani	22	474649	UB	R	2	NCM	Primi	40			✓																											
194	Ishwarya	25	473321	B	IR	1	NCM	Primi	39							✓																								
195	Santhiya	28	475922	B	R	1	NCM	Primi	38							✓																								
196	Preethi	28	472323	B	R	1	NCM	Primi	37							✓																							✓	
197	Guna Sundari	24	473455	B	R	1	NCM	Primi	38							✓																								
198	Shalni	22	476467	B	R	2	NCM	Primi	40								✓																							
199	Benazir	30	473133	B	R	2	NCM	Primi	41							✓																								
200	Vishnupriya	28	473358	B	R	1	NCM	Primi	36		✓			✓				✓										✓					✓						✓

Age	NEONATAL OUTCOME								
	Maturity			Birth Weight					
	Term	Sex	APGAR	<2.5Kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
22	✓	F	8		2.7		-	-	
23	✓	M	4		2.8		✓		✓
21	✓	M	8			3.5			
24	✓	M	8			3.5			
22		M	6	1			✓		✓
30	✓	M	7			3.25			
21	✓	F	8		2.25				
26	✓	M	8			3.25			
30	✓	F	9	2.2					
26	✓	F	8		2.8				
24	✓	F	8			3			
25	✓	M	7		2.9				
21	✓	M	8			3.2			
29	✓	M	7	2.2			✓	✓	
23	✓	F	7	2			✓	✓	
23	✓	M	7	2.3					
29	✓	M	8			2.75			
22	✓	M	8		2.5				
27	✓	M	9		2.6				
29	✓	M	8			3			
21	✓	F	9	2.4					
28	✓	M	8		2.6				
27	✓	F	8			3.6			
30		F	7	2.1					
25	✓	F	8		2.7				
24	✓	M	7		2.6		✓	✓	
32		-							
22	✓	F	8			3.4			
24	✓	F	9	2.4					
24	✓	F	8			3.4			
27	✓	F	7		2.7				
25	✓	M	8			3			
26	✓	F	8		2.9				

Age	NEONATAL OUTCOME								
	Maturity	Sex	APGAR	Birth Weight			NICU Admission	Discharged	Neontal death
	Term			<2.5Kg	2.5-3 (kg)	>3kg			
30	✓	M	8			3.1			
35	✓	M	8		2.7				
22	✓	M	9		2.5				
29	✓	F	8			3.5			
27	✓	M	8		2.6				
25	✓	M	9		2.6				
25	✓	F	6			3	✓	✓	
26		F	6	1			✓		✓
24	✓	M	7		2.5		✓	✓	
22	✓	M	8		2.7				
24	✓	M	7	1.9			✓	✓	
28	✓	F	7		2.5		✓	✓	
26		M	7	2.2			✓	✓	
27		M	8	2.3					
24	✓	F	8			3.2			
25	✓	M	9			3			
23	✓	M	8		2.8				
26		-							
26	✓	F	-	2.2					✓
31	✓	M	8		2.8				
27	✓	M	8		2.5				
37	✓	F	8		2.9				
27	✓	F	7			3			
27	✓	M	8		2.8				
23	✓	M	8		2.9				
25	✓	M	9		2.6				
26	✓	F	8		2.5		✓	✓	
23	✓	M	8		2.5				
27									
26	✓	F	9		2.9				
26	✓	F	8		2.7				
25	✓	M	8		2.6				
27	✓	F	8		2.5				
28	✓	M	8		2.8				
24	✓	F	8			3			
24	✓	M	7			3.2			
25	✓	M	8		2.8				
27	✓	F	9		2.8				
27		-							

Age	NEONATAL OUTCOME								
	Maturity			Birth Weight					
	Term	Sex	APGAR	<2.5Kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
25		M	7	1.8			✓	✓	
29	✓	M	8		2.7				
25	✓	M	8			3.4			
24		F	7		2.7				
25		-							
27	✓	M	8		2.9				
26	✓	F	8		2.6				
25	✓	F	7		2.6		✓	✓	
25	✓	M	8	2.4			✓	✓	
27	✓	M	6	2					
24	✓	M	8		2.8				
25		F	5	800g			✓		✓
26	✓	M	7		2.8				
32		F	8		2.9				
30	✓	M	9			3			
25	✓	M	8		2.9				
25	✓	M	8		2.7				
25	✓	M	7	2			✓	✓	
27		-							
27	✓	F	8		2.8				
25	✓	M	8		2.9				
23	✓	M	8			3.2			
33	✓	M	8		2.6				
25	✓	M	7		2.7				
27	✓	F	8		2.9				
29	✓	F	8		2.8				
28	✓	M	8		2.9				
28		F	7			3.2	✓	✓	
27		M	7	2.1			✓	✓	
34	✓	F	8		2.8				
33		M	8		2.5				
24	✓	M	8			3.2			
27	✓	F	8		2.8				
27	✓	M	9			2.5			
28		M	-	600g					
27	✓	F	8		2.8				
30	✓	M	8		2.9				
26	✓	F	8		2.5				
25	✓	F	7		2.4				

	NEONATAL OUTCOME								
	Maturity			Birth Weight					
Age	Term	Sex	APGAR	<2.5Kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
20	✓	M	8			3			
28	✓	F	8			3.2			
20	✓	F	8		2.9				
20	✓	M	8		2.8				
25	✓	F	9		2.8				
28		M	7	1.9			✓	✓	
30	✓	M	8		2.8				
27		M	8		2.9				
28	✓	F	7			3.4	✓	✓	
26	✓	M	9			3			
25		-							
29	✓	F	8		2.8				
27		M	6	1			✓		✓
30	✓	M	8		2.7				
32	✓	F	8			3			
28	✓	M	8		2.9				
27	✓	M	8		2.9				
27	✓	F	8		2.8				
20	✓	F	8		2.5				
25	✓	M	8		2.9				
29	✓	M	8	2.4			✓	✓	
27		F	7	2.2			✓	✓	
24	✓	M	8		2.8				
26	✓	M	8		2.5				
26	✓	F	8		2.4				
28	✓	M	9		2.9				
25	✓	M	7			3.5			
27		F	-	400g					
24	✓	F	8		2.8				
26	✓	M	8		2.9				
27	✓	M	9		2.8				
29	✓	F	8		2.5				
30	✓	M	7		2.8		✓	✓	
25	✓	M	8		2.9				
26	✓	M	9		2.8				
22	✓	F	8		2.6				
25	✓	M	7		2.5				
27		F		100g					

Age	NEONATAL OUTCOME								
	Maturity	Sex	APGAR	Birth Weight			NICU Admission	Discharged	Neontal death
	Term			<2.5Kg	2.5-3 (kg)	>3kg			
30	✓	F	8		2.9				
28	✓	F	8		2.6				
22		F	7	2.1			✓	✓	
25	✓	M	8	2.4					
28	✓	M			2.8				
30	✓	M	8			3			
24	✓	F	8		2.5				
29	✓	F	7		2.8				
29	✓	M	8		2.9				
27	✓	M	9		2.5				
27	✓	M	8	2.4					
29		F	5	1.1			✓		✓
27	✓	M	8		2.9				
26	✓	M	7			3.3			

Age	NEONATAL OUTCOME								
	Maturity			Birth Weight					
	Term	Sex	APGAR	<2.5Kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
25	✓	M	8			3			
20	✓	F	7			3.8	✓	✓	
33	✓	F	7		2.8		✓	✓	
24	✓	M	9		2.7				
26	✓	M	8			3.2			
28	✓	F	8		2.9				
27	✓	F	8		2.5				
25	✓	M	8			3.4			
29		F	7	2.1			✓	✓	
24	✓	M	9			3.2			
20	✓	F	8			3			
28		M	8		2.7				
28	✓	F	8		2.6				
27	✓	M	7		2.9				
28	✓	F	9			3.2			
26	✓	M	8		2.6				
25	✓	M	8		2.5				
25		F	-	200g					
23	✓	M	9		2.8				
28		-							
27	✓	M	8		2.6				
30	✓	F	8		2.5				
25	✓	F	8		2.8				
24	✓	F	8		2.5				
27	✓	M	6			3.3	✓	✓	
27	✓	F	9		2.7				
29	✓	M	9			3.2			
25		-							
30	✓	M	8			3			
22	✓	M	8		2.8		✓	✓	
25	✓	F	8		2.9				
28	✓	M	8		2.8				
28		M	8			3.1			
24	✓	F	9			3			
22	✓	M	7			3.5	✓	✓	
30	✓	F	8		2.8				
28		M	6	2			✓	✓	

S.No	Name	Age	IPNo	Booking Status	Menstrual Histroy	MARITAL H/O (YEARS)	CONSANGUINITY	Obstetric H/o	Gestational Age (Weeks)	OBSTETRIC OUTCOME						MODE OF DELIVERY																			CO
																			INDICATION FOR ISCS																	
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/ Abnormal Doppler	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHTT				
1	Aarthi	26	465985	B	R	2	NCM	G_3A_2	29		✓					✓																			
2	Rekha	26	465980	B	IR	2	NCM	G_3A_2	39							✓																			
3	Renuka	27	466441	UB	R	3	CM	G_3A_2	29									✓	✓																
4	Amudhavalli	31	466860	B	R	3	NCM	G_3A_2	40									✓																✓	
5	Jeyaseela	38	466404	B	R	2	NCM	G_4A_3	40		✓							✓															✓		
6	Umarani	27	466840	B	IR	2	NCM	G_3A_2	40									✓				✓												
7	Revathy	27	462578	B	R	3	NCM	G_3A_2	11	✓																								
8	Jeyalakshmi	23	467108	B	IR	2	CM	G_3A_2	39							✓																		
9	Karhini	25	467525	B	R	4	CM	G_3A_2	39							✓																		
10	Amalarani	26	466649	B	IR	5	NCM	G_5A_4	38					✓		✓																		
11	Angalarkanni	26	468417	B	R	2	CM	G_3A_2	34		✓					✓	✓																	
12	Baryrani	27	468334	B	R	2	NCM	G_3A_2	38							✓																		
13	Priyanka	26	472234	UB	IR	2	NCM	G_3A_2	9	✓																								
14	Bama	23	468641	B	R	3	NCM	G_3A_2	41									✓		✓														

S.No	Name	Age	IPNo	Booking Status	Menstrual Histroy	MARITAL H/O (YEARS)	CONSANGUINITY	Obstetric H/o	Gestational Age (Weeks)	OBSTETRIC OUTCOME						MODE OF DELIVERY																				CO
																			INDICATION FOR ISCS																		
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/ Abnormal Doppler	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHTT	
15	Madhavi	27	468311	B	R	3	NCM	G_3A_2	34		✓						✓																			
16	Lavanya	27	469114	B	IR	4	NCM	G_3A_2	35		✓						✓																			
17	Sumathy	22	474884	UB	R	4	NCM	G_3A_2	22		✓		✓		✓												✓									
18	Nalini	23	469267	B	R	3	CM	G_3A_2	39			✓						✓			✓															
19	Devi	29	468663	UB	R	2	NCM	G_3A_2	36		✓					✓																				
20	Anitha	29	469489	B	R	4	CM	G_3A_2	37					✓				✓								✓										
21	Jeilaravi	29	469804	UB	R	6	NCM	G_3A_2	39																											
22	Anbuselvi	31	469676	B	IR	7	NCM	G_3A_2	37									✓																✓		
23	Sarala	31	474985	UB	R	5	NCM	G_3A_2	14	✓																										
24	Kavitha	32	469972	B	R	4	NCM	G_3A_2	38			✓				✓																				
25	Suganya	21	470264	B	IR	3	CM	G_3A_2	35									✓																		
26	Kannagi	28	469878	B	R	3	NCM	G_3A_2	38					✓				✓																		✓
27	Pandiselvi	25	470500	B	IR	4	NCM	G_3A_2	38									✓					✓												✓
28	Chithra	22	477917	UB	R	4	NCM	G_3A_2	16	✓																										
29	Santhiya	29	471008	B	R	2	NCM	G_3A_2	36		✓	✓				✓																				
30	Anushiya	21	471058	UB	R	3	NCM	G_3A_2	39									✓					✓												✓
31	Berllin Bemita	22	471273	B	IR	2	NCM	G_3A_2	38			✓						✓			✓															
32	Manjula	23	471430	B	R	3	NCM	G_3A_2	38			✓						✓			✓															
33	Jasmin Meilda	28	471368	UB	R	4	CM	G_3A_2	40									✓				✓													✓
34	Aruna	24	472161	B	R	2	CM	G_3A_2	36		✓					✓																			

S.No	Name	Age	IPNo	Booking Status	Menstrual Histroy	MARITAL H/O (YEARS)	CONSANGUINITY	Obstetric H/o	Gestational Age (Weeks)	OBSTETRIC OUTCOME						MODE OF DELIVERY																			CO
																			INDICATION FOR ISCS																		
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/ Abnormal Doppler	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHTT	
35	Ponnumani	23	472404	B	R	3	NCM	G_3A_2	38							✓																			
36	Shanthi	28	473162	B	R	5	NCM	G_3A_2	37									✓															✓		
37	kolanjyamual	27	477917	UB	R	8	CM	G_9A_8	8	✓																									
38	Nadhiya	30	472133	UB	R	3	NCM	G_4A_3	38							✓																			
39	Sasikala	26	473639	B	R	3	NCM	G_5A_4	38									✓		✓															
40	Sheela Mary	30	473747	B	IR	2	NCM	G_3A_2	39																										
41	Rubini	26	474481	UB	R	3	NCM	G_4A_3	38			✓						✓			✓														
42	Deepita	25	475486	B	R	2	NCM	G_3A_2	38							✓																			
43	Manjula	20	475581	B	R	2	NCM	G_3A_2	39							✓																			
44	Devika	28	475578	UB	IR	3	NCM	G_4A_3	40									✓																	
45	Gayathri	20	475620	B	R	2	CM	G_4A_3	39							✓																			
46	Shanthi	20	475838	B	IR	2	NCM	G_3A_2	40									✓															✓		
47	Rajesh ari	25	475406	UB	R	2	NCM	G_3A_2	40			✓						✓															✓		
48	Usharani	28	476192	B	R	3	CM	G_3A_2	38							✓																			
49	Senganval	30	478219	UB	R	7	NCM	G_3A_2	7	✓																									
50	Saranya	27	474900	B		3	NCM	G_4A_3	38									✓				✓													✓
51	Kaliyamal	28	473591	UB	R	4	NCM	G_4A_3	34		✓				✓			✓																	
52	Priya	23	473823	B	IR	3	NCM	G_3A_2	37							✓																			
53	Amalarani	24	478204	UB	R	4	NCM	G_4A_3	20						✓																				
54	Thirumalai selvi	23	467902	B	IR	3	NCM	G_3A_2	40							✓																			
55	Vasumathy	28	468015	UB	R	2	NCM	G_3A_2	28		✓							✓																	✓
56	Menaka	24	469910+	UB	R	3	NCM	G_3A_2	26	✓																									
57	Priyanka	21	451968	B	R	3	CM	G_4A_3	37							✓																			
58	kokila	25	451827	B	R	2	NCM	G_3A_2	37									✓	✓																✓
59	Bhuvanesh ari	28	451412	B	R	3	NCM	G_4A_3	40							✓																			
60	Prema	20	401318	B	R	2	NCM	G_4A_3	38							✓																			
61	Sumathy	22	451396	B	R	3	NCM	G_3A_2	40									✓	✓																
62	Lavanya	23	451010	UB	IR	2	NCM	G_4A_3	36					✓				✓								✓									
63	Jenitta	24	450499	B	R	3	CM	G_4A_3	39							✓																			

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																			INDICATION FOR ISCS																		
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/ Abnormal Doppler	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHIT	
64	Tamilselvi	26	450332	B	R	5	NCM	G_3A_2	38 6d									✓																		
65	Suganya	24	450233	B	R	4	NCM	G_3A_2	39			✓						✓			✓																
66	Hemalatha	30	449964	B	IR	3	NCM	G_4A_3	40									✓				✓														✓
67	Suriya	25	449526	B	R	2	NCM	G_4A_3	39									✓	✓																	
68	Kanmani	25	449533	B	R	4	NCM	G_3A_2	40 5d									✓		✓																
69	Jegadeesh ari	22	449536	B	R	2	NCM	G_3A_2	40									✓				✓														✓
70	Getsiyal	22	449007	UB	R	6	NCM	G_3A_2	37 2d					✓				✓														✓			
71	Mariyammal	25	448498	B	R	5	NCM	G_3A_2	38 4d																										
72	Usha	35	442427	B	R	4	NCM	G_3A_2	39									✓														✓		
73	Muthulakshmi	27	446929	B	R	2	NCM	G_3A_2	38 5d									✓				✓												
74	Sathiya	23	407078	B	R	3	NCM	G_3A_2	40							✓																		
75	Sasikala	30	446910	B	IR	2	NCM	G_3A_2	38									✓				✓												✓
76	Mahes ari	29	446278	B	R	2	NCM	G_3A_2	35									✓							✓									
77	Suganya	23	446757	B	R	4	NCM	G_3A_2	39									✓														✓		
78	Thamaraiselvi	23	446646	B	R	2	NCM	G_3A_2	39							✓																		
79	Selvi	30	446435	B	R	3	NCM	G_3A_2	37							✓																		
80	Shivashakthi	27	446248	B	R	5	NCM	G_3A_2	39									✓	✓															
81	Revathy	31	445952	B	R	8	NCM	G_3A_2	40									✓														✓		
82	Muthulakshmi	29	448126	B	R	6	NCM	G_4A_3	33		✓					✓																		
83	Kaveri	28	451241	UB	IR	3	NCM	G_4A_3	38				✓					✓									✓							
84	Kasiyammal	27	449260	UB	R	2	NCM	G_3A_2	11	✓																								
85	Saroja	24	441701	B	R	1	CM	G_3A_2	38			✓				✓																		✓
86	Poovazhagi	29	441812	B	R	4	NCM	G_3A_2	29					✓																				
87	Ganathy	30	446104	B	IR	3	NCM	G_4A_3	38									✓								✓								✓
88	Sivaranjani	32	448471	B	IR	7	CM	G_6A_5	36		✓					✓																		
89	Savithri	22	449042	B	R	2	CM	G_3A_2	37			✓						✓			✓													
90	Bharathi	25	451012	UB	R	2	NCM	G_3A_2	40							✓																		

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										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/ Abnormal Doppler	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHTT					
91	Mahathi	25	454404	B	R	2	CM	G_3A_2	10	✓																										
92	Ezhilarasi	24	453721	UB	IR	3	CM	G_3A_2	35		✓					✓																				
93	Keerthiga	28	453810	B	R	4	CM	G_3A_2	39			✓						✓			✓														
94	Roja	27	457410	B	R	3	CM	G_4A_3	39							✓																				
95	Tamilarasi	27	456892	B	R	1	CM	G_3A_2	26	✓																										
96	Bhuvanesh ari	25	454471	B	R	2	NCM	G_3A_2	38			✓				✓																				
97	Ambika	26	447409	UB	R	3	NCM	G_3A_2	37				✓					✓													✓					✓
98	Latha	23	452172	UB	IR	2	NCM	G_3A_2	39							✓																				
99	Suraya Priyadharshini	22	451404	B	IR	1	NCM	G_3A_2	38							✓																				
100	Anuradha	24	461289	B	R	2	NCM	G_3A_2	40	✓								✓																		
101	Gayathri	28	461742	UB	R	2	NCM	G_3A_2	38							✓																				
102	Renganayagi	25	465066	B	IR	2	CM	G_3A_2	39							✓																				
103	Archana	27	461414	B	R	3	NCM	G_3A_2	30		✓							✓																		
104	Kumudhavalli	24	462525	B	R	3	NCM	G_3A_2	37							✓																				
105	Poojakumari	22	460777	B	R	4	NCM	G_3A_2	9	✓																										
106	Nivedhita	29	469786	UB	R	4	CM	G_3A_2	37									✓																		
107	Kalpana	22	466863	B	R	3	NCM	G_4A_3	11	✓			✓																							
108	Ragini	34	466421	B	IR	3	NCM	G_3A_2	38							✓																				
109	lallitha	25	467645	UB	R	2	NCM	G_3A_2	39			✓				✓																				
110	Neelavathy	27	462314	B	R	2	NCM	G_3A_2	38									✓																		
111	Rosy	25	465613	B	R	2	NCM	G_3A_2	39								✓																				
112	Kaladevi	25	466840	B	R	3	CM	G_3A_2	38									✓				✓														
113	Vasantha	24	467356	UB	IR	5	CM	G_3A_2	28		✓		✓		✓			✓																		
114	Dhanalakshmi	29	468115	B	R	7	NCM	G_4A_3	39							✓																				
115	Poongodi	25	469562	B	R	3	NCM	G_3A_2	39							✓																				
116	Lakshmipriya	26	463100	B	R	2	NCM	G_3A_2	27	✓																										
117	kousalya	20	464567	B	R	2	NCM	G_4A_3	38			✓						✓							✓											
118	Pavithra	23	461214	B	R	3	NCM	G_3A_2	38							✓																				
119	Poovizhi	25	462501	B	R	3	NCM	G_3A_2	39							✓																				

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										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/ Abnormal Doppler	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHIT
120	Chithra	25	466145	UB	R	4	NCM	G_3A_2	38					✓				✓								✓										✓
121	Selvarani	24	464512	B	R	2	CM	G_3A_2	36		✓					✓																				
122	Go ri	26	468166	B	R	2	NCM	G_3A_2	38			✓						✓																		
123	Gracy	25	469111	UB	IR	2	CM	G_5A_4	38									✓	✓																	
124	Akhila	24	468782	B	R	2	NCM	G_4A_3	39			✓				✓																				
125	Pushparani	27	467645	B	R	3	NCM	G_3A_2	21	✓																										
126	Savithiri	26	477113	B	R	2	NCM	G_3A_2	37			✓						✓	✓																	
127	Thilagavathy	25	467220	B	R	3	CM	G_3A_2	7	✓																										
128	Anjali	25	468551	UB	R	3	NCM	G_3A_2	38					✓				✓																		
129	Eshwari	30	468621	UB	IR	5	NCM	G_3A_2	37							✓																				✓
130	Stellarani	24	469001	B	R	3	CM	G_3A_2	38							✓																				
131	Radha	28	469114	UB	R	3	NCM	G_3A_2	38			✓						✓				✓														
132	Indira	27	469008	B	R	4	NCM	G_4A_3	39			✓						✓				✓														
133	Tharapriya	22	467251	B	R	6	NCM	G_4A_3	40									✓																		
134	Maheswari	25	462075	B	R	3	NCM	G_3A_2	19	✓																										
135	Bakiya Lakshmi	25	467687	B	R	3	NCM	G_3A_2	38							✓																				
136	Umarani	26	466242	B	R	3	NCM	G_3A_2	39							✓																				
137	Sasikala	27	465020	B	R	3	NCM	G_3A_2	34		✓	✓				✓																				
138	Amudha	25	465322	B	R	2	NCM	G_3A_2	32		✓					✓																				
139	Bhavani	20	464351	UB	R	3	NCM	G_3A_2	38									✓					✓												✓
140	Sumathi	22	469915	B	R	3	NCM	G_3A_2	38							✓																				
141	Malathi	25	460053	B	R	2	NCM	G_3A_2	39							✓																				
142	Deepa	24	467714	B	IR	4	CM	G_3A_2	39									✓	✓																	
143	Rajakumari	27	468466	B	R	2	NCM	G_3A_2	11	✓																										
144	Senthamailselvi	30	461940	B	IR	2	NCM	G_3A_2	35		✓			✓				✓									✓									✓
145	Sundharvalli	27	463825	B	R	2	NCM	G_3A_2	38			✓						✓	✓																	
146	Babu shalini	27	462111	B	R	2	NCM	G_3A_2	37		✓					✓																				
147	Subasri	25	460807	B	IR	3	NCM	G_3A_2	37									✓					✓												
148	Aarthi	24	462085	B	R	3	NCM	G_4A_3	40							✓																				
149	Saratha devi	25	467667	B	R	4	NCM	G_3A_2	14	✓																										
150	Prema	28	467828	UB	R	10	NCM	G_3A_2	35		✓							✓																✓		

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151	Regina	25	468826	B	IR	3	CM	G_3A_2	38							✓																				
152	Banumathi	30	464577	UB	R	2	CM	G_3A_2	39							✓																				
153	Devika	32	469939	B	R	2	NCM	G_3A_2	40									✓		✓																
154	Deivarani	24	467787	B	R	2	NCM	G_3A_2	37									✓																		✓
155	Usha	25	469004	B	R	3	NCM	G_3A_2	40							✓																				
156	Rathna malar	25	460932	B	R	3	NCM	G_3A_2	39			✓						✓			✓															
157	Vanitha	27	464478	B	R	3	CM	G_3A_2	23	✓																									
158	Angayarkanni	26	469116	UB	R	2	NCM	G_3A_2	40							✓																				
159	Ramya	25	467632	B	IR	2	NCM	G_3A_2	37									✓				✓														
160	Shanthi	24	466305	UB	R	2	NCM	G_3A_2	36		✓							✓															✓			
161	Sugapriya	20	461360	B	R	2	NCM	G_3A_2	39							✓																				
162	Jothi lakshmi	30	461092	B	R	2	CM	G_3A_2	40							✓																				
163	Jeya	24	461010	B	R	2	NCM	G_3A_2	9	✓																										
164	Malarkodi	27	460574	B	IR	3	NCM	G_3A_2	39			✓						✓	✓																	
165	Valliyammai	29	463484	B	R	2	NCM	G_3A_2	38									✓																		
166	Hema	30	462899	B	R	2	NCM	G_3A_2	38			✓						✓		✓																
167	Geetha	22	464358	B	R	2	NCM	G_3A_2	40							✓																				
168	Kalaivani	28	468951	B	R	2	CM	G_3A_2	39									✓																	✓	
169	Gayathri	27	468471	B	IR	2	NCM	G_3A_2	38							✓																				
170	Savithri	30	469417	UB	R	3	NCM	G_4A_3	37			✓						✓																	✓	
171	Bhuvana	28	468432	B	R	3	NCM	G_3A_2	9	✓																										
172	Nandhini	32	467104	B	IR	2	NCM	G_3A_2	37							✓																				
173	Fathima Beevi	33	466876	B	R	3	CM	G_3A_2	23	✓					✓																					
174	Rasiya	27	466680	B	R	2	NCM	G_3A_2	38									✓				✓														✓
175	Nadhiya	26	467029	B	R	4	NCM	G_3A_2	33		✓		✓					✓										✓								
176	Sahvithri	28	471280	UB	R	8	NCM	G_3A_2	38									✓																	✓	
177	Priyanka	30	471843	UB	R	2	CM	G_3A_2	37									✓												✓						
178	Muthulakshmi	27	471984	B	R	2	NCM	G_4A_3	39					✓		✓																				

S.No	Name	Age	IPNo	Booking Status	Menstrual Histroy	MARITAL H/O (YEARS)	CONSANGUINITY	Obstetric H/o	Gestational Age (Weeks)	OBSTETRIC OUTCOME						MODE OF DELIVERY																			CO
																			INDICATION FOR ISCS																		
										Abortion	Preterm Labour	PROM	APH	IUGR	IUD/Stillbirth	Normal Vaginal	Assisted Vaginal	LSCS	CPD	Fetal Distress	PROM/Failed Induction	GHT/Failed Induction	Postdated failed Induction	severe Oligo/Fetal Distress	IUGR/Fetal distress/ Abnormal Doppler	Placeuta previa	Abruption	Severe Preeclampsia	AP Eclampsia	Others	GHIT	
179	Jacqueline	29	472304	B	R	3	NCM	G_3A_2	35		✓					✓																			
180	Devika	25	472812	B	R	5	NCM	G_3A_2	39							✓																			
181	Priyadarshini	24	473812	UB	R	2	NCM	G_3A_2	36		✓							✓													✓				
182	Kananga	25	4733142	B	R	3	NCM	G_3A_2	38			✓				✓																			
183	Malathy	26	474312	B	R	3	NCM	G_3A_2	39							✓																			
184	Perezhil	26	474821	B	IR	2	NCM	G_3A_2	38							✓																			
185	Pavithra	27	475781	B	R	2	NCM	G_3A_2	37									✓						✓											
186	Rani	25	475820	B	R	3	NCM	G_4A_3	38			✓						✓	✓																
187	Madhumitha	23	475320	B	R	2	NCM	G_3A_2	38							✓																			
188	Gomathy	29	476812	B	R	2	NCM	G_3A_2	39							✓																			
189	Lalitha	30	476620	B	R	7	NCM	G_3A_2	38									✓		✓															
190	Pandeeshwari	25	476537	B	R	2	NCM	G_3A_2	39							✓																			
191	Rekha	27	476420	UB	R	3	NCM	G_3A_2	34		✓					✓																			
192	Kalaimathy	28	477102	B	IR	4	NCM	G_3A_2	38							✓																			✓
193	Vinothini	26	477394	B	R	5	CM	G_3A_2	38							✓																			
194	Keerthiga	26	475839	B	R	3	NCM	G_3A_2	39									✓	✓																
195	Saranya	25	478634	B	IR	2	NCM	G_3A_2	37									✓						✓											
196	Shanmugapriya	27	478332	UB	R	5	NCM	G_3A_2	36							✓																			
197	Harini	30	478012	B	R	6	NCM	G_3A_2	38							✓																			
198	Rojamalar	24	470888	B	R	4	NCM	G_3A_2	38			✓						✓			✓														
199	Vanaja	32	481253	B	R	2	NCM	G_3A_2	9	✓																									
200	Karthiga	27	480020	UB	R	5	NCM	G_3A_2	34		✓					✓																			

S.No	MPLICATIONS							NEONATAL OUTCOME									
	Antepartum							Maturity				Birth Weight					
	Severe Preeclampsia	AP Eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm	Term	Sex	Apgar Score	<2.5kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
1								✓		M	7	1.2			✓		✓
2						✓			✓	F	8			3.5	-		
3								✓		M	8			3.3	-		
4									✓	M	7			3.1	✓	✓	
5		✓							✓	M	7	1.6			✓	✓	
6	✓								✓	F	8		3		-		
7																	
8									✓	M	9		3		-		
9									✓	F	8		3]		-		
10									✓	F	8	2			✓	✓	
11								✓		M	7	1.75			✓	✓	
12										M	8		2.9				
13										M							
14										F	8		2.6		✓	✓	

S.No	MPLICATIONS							NEONATAL OUTCOME									
	Antepartum							Maturity				Birth Weight					
	Severe Preeclampsia	AP Eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm	Term	Sex	Apgar Score	<2.5kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
15										F	8	2.1			✓	✓	
16										F	8	2.3			-		
17			✓							M	-			500g	-	-	✓
18										M	7		3.2		✓	✓	-
19										M	8		2.5		-		-
20										F	7	1.75			✓	✓	
21										M	8		3.25		✓	✓	
22					✓					F F	8 7		2.5 2.6		-	-	
23										-							
24										F	8		2.6		-		-
25		✓								M	7	2.4			✓	✓	
26										F	8	2.2			✓	✓	
27						✓			✓	M	8		3		-	-	
28										-							
29								✓		F	7	2			-	-	
30									✓	F	8		2.9		-	-	
31									✓	M	8			3.4	✓	✓	
32									✓	F	8			3.5	-	-	
33									✓	F	8		2.8				
34								✓		F	9		2.5		-	-	

S.No	MPLICATIONS							NEONATAL OUTCOME									
	Antepartum							Maturity				Birth Weight					
	Severe Preeclampsia	AP Eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm	Term	Sex	Apgar Score	<2.5kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
35									✓	M	8		2.8				
36									✓	M	7			3.4	✓	✓	
37										-	-						
38									✓	F	8	2.5					
39									✓	M	8	2.5			✓	✓	
40									✓	M	8		2.75				
41									✓	F	8			3.75			
42									✓	M	9		2.7				
43									✓	F	8		2.6				
44									✓	M	9			3.75			
45									✓	M	8		2.6				
46									✓	F	7			3	✓	✓	
47									✓	M	6			3	✓	✓	
48									✓	M	7	2.2					
49										-							
50						✓			✓	M	8			3.3	-	-	
51				✓				✓		M	7	1.9			✓	✓	
52						✓			✓	F	8	2.4			-	-	
53									✓	M M	-	900g 800g					✓ ✓
54									✓	F	8	2.3					
55				✓				✓		F		900g			✓		✓
56								✓		M	6	800g					✓
57									✓	M	8		2.75				
58									✓	M	8		2.95				
59									✓	M	8			3.2			
60									✓	M	9			3.4			
61									✓	M	8		2.9				
62								✓		F	7	2.4			✓	✓	
63									✓	M	8			3.05			

S.No	MPLICATIONS							NEONATAL OUTCOME									
	Antepartum							Maturity				Birth Weight					
	Severe Preeclampsia	AP Eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm	Term	Sex	Apgar Score	<2.5kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
64									✓	M	7		2.65		✓	✓	
65							✓		✓	M	8			3			
66							✓		✓	F	8	2.2					
67									✓	M	8			3.3			
68									✓	F	7			3.6	✓	✓	
69									✓	F	7			4.1	✓	✓	
70		✓							✓	M	7	2			✓	✓	
71									✓	F	8	2.5					
72									✓	M	8			3.5			
73									✓	F	7	2.15					
74									✓	M	8		2.8				
75									✓	M	8		2.75				
76								✓		M	8	2.1			✓	✓	
77		✓							✓	M	8		2.6		✓	✓	
78									✓	M	8		2				
79									✓	M	8			3.1			
80									✓	M	8			3.5			
81									✓	F	7			3.3			
82								✓		F	7	2			✓	✓	
83			✓						✓	M	6		2.7		✓	✓	
84										-							
85							✓		✓	M	7	2.4					
86								✓		F	-	800g	-		✓		✓
87									✓	F	7	2			✓	✓	
88								✓		F	8	2.2			✓	✓	
89									✓	M	7		2.6		✓	✓	
90									✓	M	8		2.8				

S.No	MPLICATIONS							NEONATAL OUTCOME									
	Antepartum							Maturity				Birth Weight					
	Severe Preeclampsia	AP Eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm	Term	Sex	Apgar Score	<2.5kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
91									✓	-							
92								✓		M	7	1.5			✓		✓
93						✓			✓	F	8		2.9		-	-	
94									✓	M	8		2.7				
95								✓		F	7	800					✓
96									✓	M	8	2.2					
97				✓		✓			✓	F	7	2			✓	✓	
98									✓	M	8		2.7				
99							✓		✓	M	8		2.6		✓	✓	
100					✓				✓	M	8	1.9			✓	✓	
101									✓	M	8		2.8				
102									✓	F	8		2.6				
103	✓							✓		M	5	900g			✓		✓
104									✓	M	7	2.3					
105						✓				-							
106			✓						✓	M	7	2.7					
107										-							
108									✓	M	9		2.9	3.2			
109									✓	F	9			3.8			
110					✓				✓	M	8			3.3	✓	✓	
111					✓				✓	M	7						
112									✓	F	8		2.8				
113	✓			✓				✓		F	-	800g					
114									✓	M	9		2.9				
115									✓	F	8			3.4			
116									✓	M	6	800g			✓		✓
117									✓	M	8	2.4					
118									✓	F	9		3				
119									✓	F	8		2.9				

S.No	MPLICATIONS							NEONATAL OUTCOME									
	Antepartum							Maturity				Birth Weight					
	Severe Preeclampsia	AP Eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm	Term	Sex	Apgar Score	<2.5kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
120									✓	M	7	2			✓	✓	
121					✓			✓		M	7			3.2	✓	✓	
122									✓	M	7		2.6				
123									✓	M	8		2.8				
124									✓	F	7			3	✓	✓	
125								✓		F	-	500g					
126									✓	M	8		2.9				
127										-			-				
128									✓	F	7	1.8			✓	✓	
129					✓				✓	M	7			3.3			
130									✓	M	8		2.7				
131									✓	F	6		2.6		✓	✓	
132						✓				F	8		2.5				
133									✓	M	7	2.3					
134										F	-	100g					
135									✓	M	8		2.8				
136									✓	M	8			3.4			
137								✓		F	8	2.1					
138								✓		M	7	1.6			✓	✓	
139									✓	F	8	2.5					
140					✓	✓				M	7			3			
141									✓	M	8		2.8				
142									✓	M	8		2.7				
143										-							
144								✓		M		1.7			✓	✓	
145									✓	F	8		2.9				
146									✓	M	7	2.2			✓	✓	
147									✓	M	8		2.6				
148									✓	M	8		2.8				
149										-							
150	✓							✓		M	5	1.7			✓		✓

S.No	MPLICATIONS							NEONATAL OUTCOME									
	Antepartum							Maturity				Birth Weight					
	Severe Preeclampsia	AP Eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm	Term	Sex	Apgar Score	<2.5kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
151									✓	M	8		2.8				
152					✓	✓			✓	F	8			3			
153									✓	M	7		2.8				
154									✓	M	8		2.8				
155									✓	F	9			3.3			
156									✓	F	8		2.7				
157										F	-	600g					
158									✓	M	8		2.9				
159									✓	M	8		2.5				
160		✓						✓		F	6	1.8			✓	✓	
161									✓	M	8		2.9				
162									✓	M	9			3.2			
163										-							
164									✓	M	7	2.4			✓	✓	
165									✓	F	7		2.5		✓	✓	
166									✓	M	7		2.8		✓	✓	
167									✓	F	8		2.9				
168									✓	F	7			3.5	✓	✓	
169									✓	M	8	2.3					
170									✓	M	7	2.4			✓	✓	
171										-							
172						✓			✓	M	8		2.8				
173										F		300g					
174									✓	M	8	2.4					
175			✓					✓		M	7	1.9			✓	✓	
176									✓	F	8		2.9				
177	✓								✓	M	6	2.2			✓	✓	
178									✓	M	8	2.5					

S.No	MPLICATIONS							NEONATAL OUTCOME									
	Antepartum							Maturity				Birth Weight					
	Severe Preeclampsia	AP Eclampsia	Placenta Previa	Abruption	GDM	Hypothyroid	PPH	Preterm	Term	Sex	Apgar Score	<2.5kg	2.5-3 (kg)	>3kg	NICU Admission	Discharged	Neontal death
179								✓		M	8	2.3					
180									✓	F	8		2.7				
181				✓				✓		F	6	2					
182									✓	M	8		2.6		✓	✓	
183									✓	F	8			3			
184									✓	M	8	2.8					
185									✓	M	7		2.6				
186									✓	F	8		2.7				
187									✓	M	8		2.9				
188									✓	F	8		2.7				
189									✓	M	7		2.6		✓	✓	
190									✓	F	8			3			
191								✓		M	7	2			✓		✓
192									✓	M				3.2			
193									✓	F			2.7				
194									✓	M			2.8				
195									✓	F			2.6				
196								✓		M	8	2.4					
197									✓	F				3.1			
198						✓			✓	M			2.5				
199										F			2.7				
200							✓	✓		M	7	1.9			✓		✓